THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—13TH YEAR. SYDNEY: SATURDAY, JUNE 12, 1926. No. 24.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES— PAGE.
"Workers' Compensation Act: The Medico-Legal Aspect as Applied to Insurance Companies," by L. I. SKELTON 659
"Some Medical Aspects of the Workers' Compensation Act," by JOHN KENNEDY, F.R.C.S. .. 661
"Infant Feeding," by A. JEFFERIS TURNER, M.D. .. 667
"The Significance of Certain Pathological Changes in the Cerebro-Spinal Fluid," by BASIL CORKILL, M.B., B.S. 672

REPORTS OF CASES—
"Notes on a Case of Striæ Atrophicæ," by W. E. GEORGE, M.B., Ch.M. 674

REVIEWS—
Progress in Surgery 675
Advice and Help for Parents 676
A Manual on Ophthalmic Treatment 676
Radiology and the Jaws 676

LEADING ARTICLES—
A Medical Library 677

CURRENT COMMENT—
The Mechanism of "Insulin" Action 678

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—
Ophthalmology 680
Laryngology and Otology 680

BRITISH MEDICAL ASSOCIATION NEWS— PAGE.
Scientific 682
Nominations and Elections 686

MEDICAL SOCIETIES—
The Sydney Hospital Clinical Society 686

CONGRESS NOTES—
Australasian Medical Congress (British Medical Association) 686

CORRESPONDENCE—
Hospital Practice.. 687

OBITUARY—
John Brydges Mason 687
George Lockwood Laycock 687
Harold Maund Evans 687

THE MEDICAL DIRECTORY 687
PERSONAL 687
JOHN IRVINE HUNTER MEMORIAL FUND 688
MEDICAL APPOINTMENTS 688
BOOKS RECEIVED 688
MEDICAL APPOINTMENTS VACANT, ETC. 688
MEDICAL APPOINTMENTS: IMPORTANT NOTICE 688
DIARY FOR THE MONTH 688
EDITORIAL NOTICES 688

WORKERS' COMPENSATION ACT: THE MEDICO-LEGAL ASPECT AS APPLIED TO INSURANCE COMPANIES.[1]

By L. I. SKELTON,
Mercantile Mutual Fire, Accident and Marine Insurance Company, Limited, Melbourne.

ALLOW me to thank you for giving me the opportunity of addressing you on the problems confronting the underwriter and claims settler in dealing with the numerous claims made under the provisions of the *Workers' Compensation Acts* in Victoria. My remarks are directed chiefly to those members of your profession who first come in contact with the worker, and not to medical officers of companies whose further opinion and explanations are immediately available.

It is desirable, I think, at this stage, to correct the erroneous impression that the underwriting of workers' compensation risks is an extremely profitable venture. Nothing is further from the truth. For a period of two years ending December 31, 1924, the ratio of losses to premiums earned by all companies operating in Victoria was 62%. Add to this the conservative figure of 33% for expenses incurred in procuration and administration and we have a mere 5% representing profit. You might ask why competition is so keen to obtain this unpayable business. My reply to that is, that as traders must carry unprofitable lines in order to satisfy the demands of their many clients, so are companies obliged to cater for the insuring public in all departments of business or force their client to the company which is prepared to cater for his many requirements.

A correct appreciation of the fundamental principles of the Act is necessary by you, gentlemen, before you can provide the worker and the employer (and through him the insurance company) with the information which is essential to a speedy and amicable settlement of the worker's claim. To those of you who have not added a knowledge of the *Workers' Compensation Acts* to your store of knowledge on other subjects, I unhesitatingly commend a study of the Acts.

As the title implies, the Act is for the benefit of the workers and doubt in any direction leads to it being interpreted in favour of the worker. It is desirable, therefore, that in the certificates furnished by you, gentlemen, a clear statement of facts should be made. In many cases, in fact in probably the majority of arguable cases the worker

[1] Read at a meeting of the Victorian Branch of the British Medical Association on April 7, 1926.

seeks your advice with a perfectly open mind. He relates to you his symptoms, probably recites his theory as to the cause being some act in the course of his work and if his disability is consistent with some such act, a certificate to that effect at once makes him a partisan and he immediately becomes a difficult person to handle. A certificate that "Jones is suffering from strained muscles of the back caused while lifting timber" gives the worker a false sense of security quite apart from its being of little value, since the cause given is obviously assigned by the worker and cannot possibly be certified to by the doctor. Investigation on behalf of the company into the alleged accident and communication of the result to the company's medical officer may easily result in a clashing of medical opinions and our troubles begin. May I suggest, therefore, that the patient's theory be entirely disregarded from the outset and a reason sought for the disability. Should your efforts in this direction be unsuccessful, the certificate should state this fact and add that the disability is consistent with that which would follow an accident such as has been described. You have always the early opportunity of diagnosis and the company's opportunity of verifying it may be delayed many weeks through belated notification by the employer.

Many of you, gentlemen, probably hesitate to state in technical terms the nature of the disability. I cannot stress too strongly the necessity for using your own language. It is far simpler for companies to decode correctly a certificate couched in medical terms than for you to convey in simple language the nature of the patient's disability.

In practically every instance certificates contain an estimated period of disability. Whilst I hesitate rather to suggest that the estimate should be overstated than otherwise, this course is undoubtedly the better one. The worker may use the certificate as a means of enjoying an extended period of rest, but I am certain of your cooperation in our efforts to defeat the malingerer. I have in mind a case of a worker whose inability to work was certified to from time to time by one of our public hospitals, whereas I actually found him at work in a different occupation where he had been engaged for some weeks. Incidentally the discovery saved the payment of £100 as a lump sum settlement. I cite this as an instance to show that occasionally we are trapped, but only occasionally. Should the estimate be short of the actual period of disability, further certificates are demanded to the certain annoyance of patient and practitioner alike.

Might I remind you, gentlemen, that in the estimation of the period of disability regard should be had to the nature of the worker's occupation. You would, I think, be surprised, did you know the length of time that often elapses between the patient's discharge from hospital and his return to work. This is more particularly noticeable in cases of fractures. It is, therefore, desirable that some reference to the necessity for further absention from work following discharge should be made in the certificate.

Each of you, gentlemen, has of course encountered the patient whose disability is the result of accumulated acts. Such disability is known in works on the Acts as disease of gradual onset. Bursitis is the form most commonly met with, although I do not suggest of course that in all such cases the condition is the result of gradual process. Disability in these cases is not due to any act, but to a series of acts and is not regarded as an accident for which the employer is liable to pay compensation. I mention this to correct any impression that may exist that so long as the disability is caused at his work, the worker is entitled to be compensated. A great many of the claims made on employers would not be made did a correct appreciation of this fact exist. Thus in Coe *versus* Fife Coal Company, Limited (1909 S.C. 393; 46 S.L.R. 328; 2 B. 8), a miner, while engaged in letting full hutches down a steep gradient by hand, felt a sudden pain in his chest and a few days after became totally incapacitated. The arbitrator who had the assistance of a medical assessor, found that the cause of incapacity was cardiac breakdown due to the fact that the work in which he had for some days been engaged, was too heavy for him, that he was not injured by any sudden jerk, but that the repeated heavy exertion strained his heart until finally it was overstrained. It was held that the injury was not caused by an accident. Lord Kinnear, discussing the various interpretations placed upon the word "accident" by the House of Lords in Fenton *versus* Thorley, said:

> It seems to me that all these interpretations of the word point to some particular event or occurrence which may happen at an ascertainable time and which is to be distinguished from the necessary and ordinary effect upon a man's constitution of the work in which he is engaged day by day.

A section of the Act specifies certain percentages of compensation to be paid for specific injuries, such as loss of joints of fingers, loss of sight *et cetera*. Again, in the event of serious injury to the hand is the worker buoyed up with a false sense of security if a certificate includes a reference to the diminished use of the hand. From the point of view of the Act itself the employer is not concerned with this, so long as the worker is capable of earning a wage at least equal to that earned by him at the date of injury in some suitable employment. Time alone will tell whether his efficiency as a worker is impaired. I recently met with such a case in which the worker was told by his doctor that the usefulness of the hand was reduced by a considerable percentage. Fortunately the man was a "trier" and after receiving an offer of continued compensation for a couple of weeks until he gave the hand a fair trial, he was delighted to find that his earning powers were not diminished at all. Unless the injury falls within the four walls of that set out in the schedule, reference to diminished use is worthless. While on the subject of Fourth Schedule claims, I would urge your cooperation in the matter of adequately defining the loss.

Most companies have certificates in skeleton form so that the exact loss sustained may be shown

and to insure the payment of adequate compensation, this form should be marked as requested.

In order that the employer may be safeguarded by further claims by a minor, registration of the settlement of a Fourth Schedule claim with the Registrar of the County Court is necessary and the money due to the worker is paid into court for distribution as the court thinks fit. Along with the documents must go a certificate in prescribed form. Often the request for a certificate in such form is considered unwarranted but, as I have said, it is a document essential to the registration of the agreement. It should state precisely the loss sustained and also state the date on which the worker will be able to resume work. Obviously the certificate will be given in the last few days of his disability and incidentally will be paid for by the worker.

After the very concise manner in which the subject of hernia claims has been dealt with by Dr. Kennedy, I will content myself by saying that almost without exception claims for hernia give rise to more trouble than any other single condition. I am aware that medical opinion on the question of traumatic hernia is divided, but in no workers' compensation act is the position so admirably dealt with as in the *Workers' Compensation Act* of the State of New Jersey, United States of America. The section deals with the subject in this fashion:

> Hernia is a disease which ordinarily develops gradually, being very rarely the result of an accident. Where there is real traumatic hernia resulting from the application of force directly to the abdominal wall, either puncturing or tearing the wall, compensation will be allowed. All other cases will be considered as either congenital or of slow development and not compensable, being a disease rather than an accidental injury; unless conclusive proof is offered that the hernia was immediately caused by such sudden effort or severe strain that, first the descent of the hernia immediately followed the cause; second, that there were severe pains in the hernial region; third, that there was such prostration that the employee was compelled to cease work immediately; fourth, that the above facts were of such severity that the same was not noticed by the claimant and communicated to the employer within twenty-four hours after the occurrence of the hernia; fifth, that there was such physical distress that the attendance of a licensed physician was required within twenty-four hours after the occurrence of the hernia.

Am I unduly optimistic in hoping that the time is not far distant when the medical fraternity at least in Victoria will certify on these lines?

Section Fourteen of the Second Schedule of the Act as amended gives the employer and the worker definite rights in the matter of appealing against the decision of a medical practitioner as to the worker's condition or fitness for employment. Both parties may apply to the registrar of a county court for the matter to be referred to a medical referee who has been registered as such under the *Workers' Compensation Act*. His fee is fixed at not exceeding two pounds. In actual practice such a procedure is seldom adopted and it is desirable that in all cases there should be give and take and that the case in dispute should be kept out of court and referred privately to a nominated specialist whose fee need not be limited to the figure mentioned. This aspect strengthens the need for all certificates to be concise and in conformity with the requirements of the Act and the giver to be prepared for his expressed opinions to be subjected to the keenest cross-examination by a judge and by a medical referee sitting with the judge who may summon any such referee to act as an assessor. (See Section 3 of the Third Schedule.)

The term certifying medical practitioner is defined in the Act as meaning a legally qualified medical practitioner appointed under the Act by the Governor in Council or one appointed or acting as a certifying medical practitioner under Section Eleven of the *Factories and Shops Act*, 1915.

Under Section Eighteen of the Act the only person who may certify that a worker is suffering from an industrial disease and whose certificate is acceptable is a certifying medical practitioner and he must reside in the district in which the worker was employed. It would appear then that members of your Association owe it as a duty to their patients that they should be registered as certifying medical practitioners and familiarize themselves with the regulations concerning every aspect of the Act in relation to the giving of certificates. Failure to do this may result in that worker being forced to seek other advice, as he must do if he desires to prosecute his claim. The certificate as to industrial disease must conform to the regulations as laid down in *Government Gazette*, Number 79, of March 31, 1916. Here again either the employer or the worker has the right to have the matter referred to a medical referee whose decision shall be final.

When approached to address you, gentlemen, on this important subject, I confess to have been struck with the great opportunity of bringing about the complete understanding which is desirable. Having agreed to undertake the task, I felt that the matter should have been placed in abler hands than mine. Having attempted it, I feel that I have not done the subject justice. However, if I have at least paved the way to a clearer understanding of the difficulties confronting us, then some good has resulted. If in trying to conform to all the many and varied requirements of the Act, worker and employer, you sometimes feel aggrieved, you will I hope spare us a thought in our endeavour to administer the Act correctly with satisfaction to both worker and employer.

SOME MEDICAL ASPECTS OF THE WORKERS' COMPENSATION ACT.[1]

By JOHN KENNEDY, F.R.C.S. (Ireland),
Melbourne.

I WISH to thank you for the honour you have shown me in asking me to read this paper this evening and to present to you many of the problems which one meets in dealing with claims for compensation.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on April 7, 1926.

In considering these problems I think that it will be helpful to us all if I deal with the subject as I would with an actual claim and discuss it under the following four divisions: (i.) The actual disability; (ii.) the cause of the disability; (iii.) determination of the degree of disability and (iv.) treatment of the injured worker.

The Actual Disability.

To the claimant the accident, if any, is the prominent thing. He replies to the question as to what is the matter with a detailed account of what may be the accident. Even in such an obviously inflammatory condition as appendicitis or epididymitis there is a detailed account of some strain perhaps days or weeks previously. Thus the examiner is tempted to reason from the accident to the injury rather than in the more logical way from the injury and the condition present back to the accident. Hence it is most important to determine first of all in any case the nature of the condition from which the claimant is actually suffering. This is best illustrated by any of the so-called strains, particularly "strained back." Almost any condition causing pain in the back and side of the body has been certified as a strained back and an endeavour has been made to establish a claim on that basis. Apart from definite injuries such as a torn muscle or ligament with an onset immediately following definite strain come all the inflammations of muscles (fibrositis), bones, joints, nerves, kidneys (stone, pyelitis or abscess). Every one of these has been called a strained back and a claim has been made on that basis. It is quite obvious that the doctor has been misled by the sequence of events as given by his patient and his diagnosis is quite correct in 70% of cases and the condition present is one of injury due to accident; but in the remaining 30% only a careful investigation and an attempt to establish an accurate diagnosis will elucidate the true cause of the disability. The difficulty comes in picking the 30%. In the first place and for the man in general practice getting the investigation carried out I might state here that for the better paid worker there are plenty of experts ready and willing to help. The other group of persons suffering from the conditions difficult to diagnose are always regarded kindly at hospital out-patients' departments. Claims have been made on the basis of strain of some such injury for almost all the ills the flesh is heir to except certain infectious diseases and physiological conditions. Attention may be drawn to a few of the more common apart from back conditions: strained heart for advanced myocardial disease and valvular disease, pneumonia, pleurisy with effusion and pulmonary tuberculosis; strained lung for advanced pulmonary tuberculosis with hæmoptysis, no definite unusual event being assignable as a cause; strained abdomen for gastric ulcer or appendicitis; strained bladder for prostatic enlargement with hæmorrhage; strain for gonorrhœa and epididymitis or gonorrhœal adenitis in both sexes.

Hence one finds it safer to disregard the accident and in these difficult cases to examine the claimant carefully in order to determine exactly what is the matter with him and to reason back from this to the accident and thus to discover whether his condition could be the result of injury due to such accident.

The Cause of the Disability.

Once the diagnosis has been established it is quite a simple matter to reason back to its cause, but quite a different problem to establish that cause as an accident arising in and out of the claimant's work.

Under this heading it is proposed to discuss groups of cases which have arisen and the means adopted to establish a connexion with accident or otherwise.

Head Conditions.

In most head cases there is a definite connexion between the manifestation causing the disability and the trauma, but in some this is not clear. Cases in which the disability does not occur for days or weeks after the injury, are apt to cause difficulty. For the establishment of a claim it is necessary that there must definite evidence of injury due to accident, contusion, scars, X ray evidence or other signs of fracture or in cases of neurosis evidence of nerve disturbance or concussion at the time of trauma and the manifestations which appear, must be such as would follow an injury in that position.

This is well illustrated by the following history:

A man fell off a horse and resumed work after momentary concussion. Four weeks later he reported with severe headache and a gradually developing hemiplegia. The definite connexion with trauma was established by the history of a right parietal hæmatoma and later by the removal of a mass of degenerating clot from beneath the contused area.

The story is quite different when with the claimant's account of accident there is no history or evidence of injury. Then some cause other than trauma must be sought. Simple and tuberculous meningitis as well as cerebral abscess may be shown under certain conditions to be due to injury. In the face of a diseased state of the arteries or of the brain tissue injury may under certain conditions be a definite factor in the development of nerve manifestations. Such a connexion may be established in apoplexy or in general paralysis of the insane, but there must be a very definite connecting link between the trauma and the disability. In apoplexy there is immediate evidence of some brain disturbance though the onset of disability may be delayed. In general paralysis of the insane trauma severe enough to cause disability is followed soon afterwards by nerve manifestation or mental change.

A man fell off a ladder and was disabled by general contusions for some weeks during which time manifestations of general paralysis of the insane became evident. The late Dr. Beatty Smith in consultation reported that while the fall may have been due to the nerve condition, there was no evidence to that effect or of previous mental condition and it was well recognized that an injury severe enough to cause some weeks disability could precipitate the nerve change present.

Neurasthenia is common after gross injuries, but also occasionally follows relatively slight head

injuries. A blow on the vertex is apparently more prone to be followed by such results than in other parts of the skull. Two cases illustrate this condition.

One patient was struck behind the fronto-parietal suture with the edge of an iron bucket. Another was struck in the same place by a piece of stone falling down a shaft. Transient concussion ensued in both instances. Abrasions and contusions were found. No fracture was disclosed by X ray examination. There was no neurological evidence of brain disturbance. In both the initial headache continued and led to definite neurasthenic manifestations. Neither patient returned to work and after prolonged disability both claims were settled by lump sum payments.

In this connexion it may be indicated that as well as the usual signs of neurasthenia such as sweating and vasomotor changes I have observed that there is always a considerable pulse pressure with what would be for the individual a low diastolic pressure. This relationship has not been observed in hysteria or neurosis and is an important guide in prognosis. Hysteria and neurosis clear up with the payment of the claim, but neurasthenia does not.

Spinal Injuries.

Myelitis may follow concussion of the spine and is usually due to hæmorrhage into the cord. Cases which present difficulties, are those characterized by slowly developing paraplegia. A claim could be established in such cases by definite evidence of spine injury followed by nerve pain and then paralysis below the area injured.

More common are cases of Pott's disease. Three groups are recognized: (i.) A primary group without definite evidence of tuberculosis; (ii.) a secondary group with definite evidence of tuberculosis elsewhere; (iii.) quiescent Pott's disease. The first and second groups may be traumatic, if there is definite evidence of injury to the spine. For example, according to Bosanquet pain and disability definitely referable to a particular vertebra followed within a reasonable interval, such as three to six weeks, with a limit of three months by the development of Pott's disease in the vertebral lesion, would be a case in point. Claims made on a basis of accident without definite evidence of injury to a vertebra are usually rejected. Old Pott's disease may be stirred up by injury. Here symptoms and disability follow rapidly on the injury. Accident may be of the nature of a fall, a blow directly or indirectly, concussion of a vertebra, as by a fall on the buttock.

Heart Conditions.

Strained heart is a condition for which a claim is put forward every now and then. In this connexion three groups of cases are recognized.

The first group includes persons with advanced heart disease or vascular degeneration whose disability arises in the course of their ordinary work. Although they can definitely refer it to some heavy strain, careful inquiry shows that there was nothing unusual about the strain. Moreover, clinical examination shows that the failure was the natural outcome of the disease and, although accelerated by anything in the nature of laborious work, was not precipitated by the strain complained of.

In the case Spence *v.* Band and Company, quoted by B——— and L——— J——— the claimant exerted himself considerably by lifting a truck on to the rails. He felt a sharp pain and stopped working. He became permanently disabled. The claim was not allowed. Advanced heart disease of long standing was present. This was bound to manifest itself sooner or later and might do so in the absence of exertion.

Several similar cases are quoted by Jones and Llewellyn.

The second group comprises persons with very slight clinical evidence of heart disease beyond a slight degree of dilatation, but with definite signs of neurasthenia. They complain of palpitation, præcordial pain (pseudoangina), rapid breathing or breathlessness which is not true dyspnœa. All the patients seen can definitely refer their disability, usually præcordial pain, to some unusual effort. All have definite septic foci such as infected teeth or tonsils or they have had a recent attack of influenza. They are usually young men, somewhat debilitated and apart from the trauma they would be regarded as suffering from toxic myocarditis with a considerable degree of neurasthenia. Most of them come within this group and Jones and Llewellyn call their condition a cardiac neurosis. In the strict sense they are not suffering from strained hearts at all, but from traumatic cardiac neurosis.

The third group comprises those suffering from true heart strain. Rupture of the heart or of the heart valves has been known to occur as a result of violent strain, but such a case has not come within my experience. Patients with definite evidence of cardiac dilatation, with compensatory symptoms and with no signs of cardiac disease and with a clear history of strain and onset, are always regarded as suffering from heart strain. The prominent features about them are the absence of septic foci, the absence of neurasthenia and rapidity of improvement.

Pneumonia and Pleurisy.

Pneumonia and pleurisy may follow an accident. A claim may be established if the following conditions are present: (i.) Definite accident, (ii.) a localized injury to the side, (iii.) an onset delayed from two to six days with definite symptoms pointing to a lesion of the chest or lung during that period.

Hernia.

Persons claiming compensation on account of hernia may be divided broadly into two groups: (i.) those who are disabled because of definite strain or injury due to accident and (ii.) those who are disabled because they have a hernia requiring operation. In the first group there is always a history of an unusual event, an extra strain or accident immediately followed by symptoms of injury such as pain and disability. When the patient presents himself for examination, he has a painful swelling which is tender. If the delay is long enough before operation is undertaken, signs of effused blood may become evident at the external ring, evidence of the tearing of tissue when the hernial contents were forced into the canal. In other words a definite strain of the tissue has occurred. The size does

not count. A small preexisting hernia may be suddenly increased in size. More bowel *et cetera* may be forced into it by accident and disability may be caused from the definite injury produced.

In the second group there is no disability because of injury. There may be a history of lifting heavy weights or some heavy weight and of varied sensations even slight pain in the groin. The patient consults his doctor usually on account of the lump and the presence of a hernia is diagnosed. It is not painful, however, nor tender nor are there any signs of recent injury. The external ring is usually larger than normal and the hernia is easily reduced and readily reappears without pain. Careful consideration of the facts of the case will show at once that this is not a traumatic hernia. The presence of a painless swelling shows immediately that it is of slow development. The ease with which it can be reduced and its manner of reappearance without pain or tenderness confirm that. On the other hand it must be admitted that the forcing suddenly of even the smallest amount of bowel or omentum through a ring and along a canal which has never been stretched must always be associated with injury and with considerable pain. To prove this let anyone inject exactly ten cubic centimetres of fluid subcutaneously, serum for instance and note the effect. Then consider the result of forcing bowel or omentum along a very small canal and causing pressure on a very delicate plexus of nerves. The crux of the whole position is that the disability must be caused by injury due to accident and must not be the result of some manifestation due to imperfect development in the man.

In America a clause in the *Workers' Compensation Act* of the State of New Jersey is to the effect that hernia is not ordinarily the result of accident, but due to personal defect. Compensation, therefore, can be claimed for disability on account of this condition only in unusual cases, that is when there has been definite accidental injury with the corresponding pain, symptoms *et cetera*.

Lesions of the Genitalia.

Claims have been made at various times on account of conditions in the genitalia of persons of both sexes said to be the result of strain, of injury due to accident, but careful examination usually determines another cause. Claims have been made at one time and another for such conditions as chancre, epididymitis, nontraumatic orchitis, ordinary hydrocele or varicocele in man and adnexal disease, prolapse *et cetera* in women.

Injuries to the Back.

Injuries to the back probably constitute the most difficult field for any examining surgeon. Leaving aside the results of such accidents as falls or blows which are usually straightforward, it is in the realm of the "strained back" that the greatest difficulty occurs. Strained back may mean anything, being simply a pain in the back as far as the claimant is concerned, but to the examining surgeon it may be a manifestation of other things apart from a torn muscle or a ligament. It may be the first symptom of œdema of the spinal cord, of a vertebral or renal condition and may indicate the presence of anything from the simplest type of inflammation to a malignant growth. Hence a very careful investigation needs to be carried out in all "strained backs" except the simplest and most definite type of injury due to accident. Conditions in the latter group are usually very definite, the onset is sudden and associated with some unusual event in the course of work. The symptoms and signs are unilateral, are always present at the muscle attachments and are characterized by pain, tenderness, swelling and limitation of movement of the spine in the direction of stretching the muscle.

Lesions of the inflammatory type, lumbago, fibrositis or rheumatism are somewhat different. The onset is rarely sudden as in injury. It comes on, as other inflammatory conditions do, with pain which gradually increases in severity and becomes most pronounced after the claimant has cooled down at the end of the day's work and has rested. Perhaps it is not severe until he attempts to get out of bed next day. Compared with a traumatic case there is a striking difference. In one there is the sudden onset associated with an unusual event and disability for any form of strain which calls on the injured muscle. In the other there is pain in the back in the course of ordinary work, no disability for the rest of that day, but considerable exaggeration on resting, the location of the pain is also characteristic. Inflammation affects a muscle in its belly, is diffuse and usually bilateral. Strain always produces symptoms at a muscular attachment or at the point of union of muscle with tendon or aponeurosis, it is always localized to the one definite area and is unilateral The tenderness is definite and localized in strain, it is indefinite and diffuse in inflammation. In strain only those movements involving injured muscles are affected, lumbago or movements of the back are painful. In regard to the rate of progress persons suffering from strain improve rapidly, those suffering from simple muscle strain, if efficiently treated, usually resume work in two or three weeks. Persons with lumbago require five or six weeks sometimes longer before resuming work. Septic foci are always present in all inflammatory conditions and the patient's rate of progress apparently depends on the efficiency of treatment of these foci.

Other obscure causes of backache occur and as they may become manifest during the course of work and can always be referred to some special effort or may follow a relatively insignificant trauma, they are often regarded as injuries due to accidents and form the basis of a claim. Under this heading come the group of conditions characterized by preexisting disease not in any way due to injury or even occupation, but liable to manifest themselves when work is being performed by the diseased part.

This group includes commonly all forms of disease of the spine, bone or joints, simple or specific types of inflammation, even malignant disease, likewise all forms of kidney disease which may manifest themselves as pain in the back, and similarly prostatic

conditions in men and uterine displacements in women.

Spine Conditions.

Spine conditions often appear as definitely due to trauma, but the symptoms and signs are out of all proportion to the severity of the accident, or what should be the real injury, an apparently simple muscle strain with or without unusual effort or a relatively slight blow being followed by most pronounced disability. Improvement in X ray technique has shown that these spine conditions are almost always the result of osteoarthritic changes around the vertebral articulations and that the back condition is a manifestation of this disease. Whilst on the one hand it may be shown from a careful consideration of all the facts, especially those in relationship to the injury and the accident that the trauma precipitated the condition and that it is really an injury due to accident, on the other hand it must be admitted that it is a condition primarily and wholly of disease and not in any way due to work.

Kidney Conditions.

It is remarkable how persons with kidney conditions come along year after year with the small berry fruit crop, both to the out-patient clinic and as claimants for "strained back." Clinically they are suffering from definite kidney condition. They complain of sudden onset of pain which may occur during the course of work. They have tenderness in the renal angle and down along the ureter. Frequency of micturition is constant and careful examination reveals the presence of cells and crystals in the urine. The symptoms are often unilateral, but there is no limitation of the movements of the spine. Pyelitis, movable kidney and stone are occasionally used as grounds for a claim, but unless there is definite evidence of injury due to accident and unless it can be shown that the kidney condition is the result of this, the claim is disallowed.

Absence of Lesions.

The last group of "strained back" comprises those which are not affected by any lesion. A case is relegated to this group only when the most thorough and careful investigation has failed to reveal the presence of either injury or disease and when this has been confirmed by a consultation with an independent physician or surgeon. In some instances there has been a definite injury which has long since cleared up, and the condition remains as a definite neurosis.

In the case of Storey *v.* The Union Steamship Company, Limited, a man had had a fall while carrying a bag of wheat on his back. He had apparently injured his posterior sacro-iliac ligaments. Months later he was sent for examination. He stated that he was unable to flex his spine or to carry weights on his back. Sitting on a couch, he could bend his back freely in all directions. While standing up and making an attempt to bend forwards he ended by making a dive head first to the floor. Examination failed to reveal any abnormality and as he claimed 100% disability, the case was contested. He was given compensation for three months' partial disability. Two weeks later he resumed work as a wheat lumper.

Clinically this group manifests itself as a disability out of all proportion to either the degree of trauma or the clinical findings. The onset may be either sudden or gradual. The disability is usually absolute as far as the back is concerned all movements in the erect posture being involved. While standing, a man with an organic type of condition holds himself with his centre of gravity behind his hip joints; the man with a functional condition bends slightly forward with his centre of gravity in front. Tenderness is not constant in position as in trauma, but varies perhaps over the whole lumbar area. There is an absence of true muscular spasm. Movements are atypical, he bends forward directly from his hips and holds the spine stiff. A man suffering from a true injury or disease bends by pushing his buttocks back to stabilize himself on his hip joints. While sitting up he can usually be encouraged to touch his toes, flexing his spine without difficulty. Inconsistencies of certain movements also appear. For example flexion movements of the thigh against resistance are often said to be painful. Movements of extension are not so, though in this latter type the lumbar muscles are involved. Arm movements against resistance may reveal a normal *latissimus dorsi* muscle and yet pain and tenderness are located in that region of the spine.

Joint Conditions.

Ordinary traumatism of joints presents no difficulty, except that very frequently one finds a strained knee regarded as though it were affected by a simple synovitis rather than by a torn ligament or sometimes by a displaced cartilage. The subacute or chronic type of arthritis is somewhat different. A most careful clinical examination aided by good X ray plates is essential for the determination of the exact condition of the various parts of the joints; evidence of injury must be carefully sought and a history obtained of the accident and of the time and mode of onset of the joint condition. In all traumatic cases there is always a definite history of accident immediately followed by severe pain and disability at the joint. The joint gradually becomes the seat of arthritis. The following case would probably illustrate my meaning.

Two men were pulling on a rope which broke and they fell back, one across the other's right hip. Work was resumed at once, but the claimant had a slight pain in the hip and this passed off in a few weeks leaving a weakness at the hip. He remained in this condition for three months, then he became suddenly disabled from severe pain and stiffness of the hip joint. Examination revealed some flexion and adduction deformity of the limb. There was a swelling in front of the neck of the femur. There was limitation of movements of the hip in all directions. No wasting was present, but 0·6 centimetre (one-quarter of an inch) of shortening was found. X ray examination revealed slight displacement of epiphysis. The condition was one of partial separation of the epiphysis and chronic arthritis of the hip joint.

Another type of late arthritis due to trauma is that following contusion of the cartilage or in the knee of the pads of fat beneath the patella. The history is generally somewhat similar to the one following:

A girl fell while going upstairs, catching her knee against the upper step. She felt severe pain at the time, this gradually passed off, but was followed in two weeks by recurrence of the pain with signs of subacute arthritis. Definite tenderness of the front of the joint was present. On X ray examination the outline of the cartilage was seen to be blurred, a condition which Jones calls a cartilage contusion.

Industrial Diseases.

Four industrial diseases are provided for in the Victorian *Workers' Compensation Act*, namely lead, mercury, arsenic and phosphorus poisoning. Of these the first is much the more common and it will be considered somewhat in detail.

The three types of lead poisoning met with are familiar to all, the arteriosclerotic, the colic and parietic. The psychoneurotic form is much rarer. In all a definite grade of anæmia is usually present. However, as any one of these three forms may be caused by other conditions, care must be taken to exclude the presence of these conditions in making a diagnosis. The fact that a claimant is working in a lead factory and has one or other of these manifestations is not sufficient in itself to establish a diagnosis. Persons with chronic interstitial nephritis, gastric cancer, gall stones and chronic appendicitis have all been certified as suffering from plumbism. Absence of conditions such as one of these together with the definite source of lead is essential for the establishment of a claim on that basis. The presence of the blue line of lead on the gums is diagnostic, but its absence is of no value whatever. Only two out of the last ten persons suffering from lead poisoning manifested a blue line. Likewise examination of the blood for basophilia does not give the reliable results which are obtained from examination of the urine. The latter is the most reliable method of all and consists in a quantitative estimation of the lead. Anything below one to six milligrammes per litre of urine is regarded as not being due to lead poisoning. Such a quantity would be yielded by anyone exposed to lead contamination apart from poisoning. In all cases of plumbism larger quantities are found. This test is carried out for me at the Department of Chemistry, Melbourne University. It costs a few guineas, but the information gained is of such great value as to render that negligible. Every person with a low quantity has ultimately turned out to be suffering from some condition other than lead poisoning, whilst in doubtful cases with a high quantity the condition has been shown ultimately to be due to lead poisoning.

Determination of the Degree of Disability.

The degrees of disability are permanent or temporary, total or partial or none at all. Total permanent types are definitely defined in the Fourth Schedule of the Act. Such conditions as fracture of the spine with contusion of the cord or total blindness need no comment. Similarly no comment is called for in regard to the person who, though injured, has not been disabled for his class of work. Such injury includes superficial wounds or contusion in parts of the body such as the face which are not used in the course of work. This group also includes malingerers when proved to be such. It may be well to note that less than 3% of claimants are malingerers. It should, however, be considered that men are disabled by conditions such as laceration of the hand *et cetera* after healing, with which they would have worked in precompensation days. The group of temporarily totally or partially disabled persons includes the majority of the claimants and great care and discrimination have to be exercised in dealing with them. This is well illustrated by two groups of cases. The traumatic neurasthenic requires rest and treatment and is totally disabled, but the traumatic neurotic requires work and exercise and is partially disabled. This advice is not given in order to save the insurance company as is often thought, but I think it is generally recognized that the sooner the neurotic gets back to work, the sooner he will get well. In like manner when repair has been effected, most persons suffering from injury to bones or joints improve more rapidly once they can get back to work, even if partially disabled. There is a tendency to regard the wasted muscles as reason for further total disability, but one must not forget that these muscles will recover only when given the work which originally developed them.

Permanent partial disability can be determined only by a knowledge of the claimant's work and of the exact amount of efficiency possible in the injured limb. The great majority of these cases comprise injuries in the neighbourhood of joints, such as incompletely reduced Colles's or Pott's fracture. A badly reduced Pott's fracture will always leave from 15% to 30% loss of efficiency in the limb. Yet the most hopeless looking fracture in the middle of the shaft of the tibia with good alignment may conclude with a 100% efficiency of the limb. A severe crushing of the hand with fracture of several metacarpal bones may in the end be much superior for the claimant's class of work than a simple laceration involving tendons with loss of gripping power of the fingers. Considering injuries in general terms, it must be recognized that while surgery can repair a damaged limb, it cannot restore a destroyed part such as the lens of an eye, a joint or part of a limb. Whilst many of these cases are provided for in the Fourth Schedule of the Act, many of them, although resulting in diminished earning capacity, are left to the discretion of examining medical officers. Hence as soon as the limits of repair are reached or as soon as these can be assessed, it is well to consider the worker's loss, avoid further depletion of his lump sum for settlement and send him back to work to make the best of his loss. It is surprising in many cases to see what a best that can be, even with malunited fracture or adherent tendons. Eye conditions are difficult and are left to eye specialists. Limb conditions are usually assessed in terms of percentage loss of the particular limb under the Fourth Schedule.

Treatment of the Injured Worker.

Treatment of the injured worker had best be considered under two headings: (i.) Primary treat-

ment and repair of the injury; (ii.) reeducation of muscles and joints and rehabilitation of the injured worker. In regard to the first of these the efficient manner in which treatment and repair are carried out at our hospitals, leaves little to be desired and naturally sets a very high standard for the man in general practice. There are, however, a few suggestions that I would like to make to him. In the first place he should make the fullest possible use of modern methods of investigation, especially X ray examination. Expense should not stand in his way for this, since a note to any out-patient surgeon will secure investigation of the condition and advice and help regarding treatment.

It is difficult to name one bone in which fracture has not been treated as a sprain or contusion until discovered weeks or months after the injury, when an opinion has been sought to explain the prolonged disability. All the bones of the limbs, pelvis, spine and skull come within this list. In some conditions such a crack through a bone without displacement, no harm results, but in conditions such as an unrecognized Colles's or Pott's fracture or fracture of the neck of the femur, the outlook for the man is much more serious and very considerable expense is incurred by the insurance company. In these cases the man is permanently crippled and never again resumes laborious work. The method of dealing with these cases is simple and has frequently been demonstrated to members of the Association. In regard to sprained joints I would state that as a rule they are regarded too lightly. Sprained ankle is usually treated by the old-fashioned slipper and bandage, a method condemned by surgeons many years ago, instead of by fixation with strapping or plaster. Sprained knees are usually certified as traumatic synovitis and are treated with Scott's dressing and a bandage. This is probably an excellent *placebo* for the patient, but is not of much value when there is a torn ligament and occasionally an unreduced cartilage displacement. A man suffering from a sprained ankle joint should be fit to resume work in a proper boot in three weeks and if suffering from knee joint displacement, in six weeks, but in actual practice this time is usually two or three times as long.

Reeducation and Rehabilitation.

I was very much impressed during my recent visit to America by the extent to which reeducation and rehabilitation are carried out in various industrial centres. The organization is financed by the State, organized by a committee which includes some of the leading orthopædic surgeons in the State and run by a paid staff of doctors, masseurs *et cetera*. Every imaginable physical therapeutic device is apparently used such as Zander's machines of all types to exercise not only every joint but almost every group of muscles in the body. One large group of machines is driven by power and is capable of remarkably fine adjustment and regulation, the other is driven by the patient himself. The patients are sent to this centre from the various hospitals and by private practitioners after primary treatment and repair of the injury. I was not able to get definite figures regarding shortened disability, but judging by the cost of the highly paid staff and expensive plant on the one hand and our knowledge of the industrial American on the other, the centre must show a profit. The question will naturally be asked as to why such a centre cannot be carried out here. For the last few years I have run such a centre in a small way in connexion with one of the larger insurance companies. This centre when running full showed a very definite profit in the way of compensation to the company and a much greater one to the injured worker in the shortened period of disability and in the wages earned.

To establish large centres equipped and run on the American plan would entail considerable expense. That should be no bar, provided it can definitely bring about a shortened period of disability to the injured worker. The economic saving in getting these efficient workers back to the industry would more than counterbalance the loss in their increased production.

Conclusion.

Finally why should not an industry support its wastage? The recent war supported its own wastage and application was not made to the public charity hospitals for free treatment. Hence is there any reason whatever why modern industry should require charity to repair its wastage? Our out-patient departments are full and our massage centres are loaded with persons receiving charity treatment for the effects of industrial accident. Has a single industrial organization paid adequate remuneration for the repair of its wastage over the period of the last ten years?

INFANT FEEDING.[1]

By A. JEFFERIS TURNER, M.D.,
Brisbane.

I PROPOSE to discuss with you tonight a subject of every day practice, a subject of which everyone knows something and no one knows everything. Simple at first sight, it grows more difficult the more one considers it; indeed it bristles with controversies and yet a degree of knowledge and experience extremely useful for practical purposes can be attained by any practitioner who will patiently observe clinical facts in a scientific spirit. Personally the more knowledge and experience I have of infant feeding, the less inclined I am to talk about it. And yet one must talk about it, unless one consents to leave the subject altogether to the arm-chair theorist, the commercial advertiser and the irresponsible person. For these never cease to ventilate their ignorance. Every young mother is continually, even daily, receiving bad advice from the

[1] Read before the Ipswich Medical Society on December 17, 1925.

advertising columns of newspapers and magazines, from her relatives, friends and chance acquaintances, from midwifery and other nurses and sometimes, alas! even from her medical attendant. We must not therefore omit any opportunity of qualifying ourselves to give her better advice nor refrain from doing so. Moreover, we should do all in our power to assist the efforts of the nurses trained in our infant clinics to disseminate simple truths which are unknown or forgotten, and to discredit foolish traditions which are powerful for evil, for in this way only can we hope to diminish our present high infantile mortality which depends more than anything on the aggressive ignorance of uninformed or thoughtless though perhaps well-meaning people.

The first step to obtaining a real knowledge of infant nutrition is to obtain a good weighing machine accurate to fifteen grammes (half an ounce) and to do one's weighing oneself. In any difficult case it is of inestimable value to know whether the infant is gaining or losing weight. To rely on vague general impressions is to navigate a stormy sea without a compass. Secondly you must learn how to observe your infant, both with your own eyes during the brief periods for which he is before you and through his mother's eyes (making due allowance for her prejudices and emotions) during the long periods when he is not before you. Is he happy and contented? Is he hungry and discontented or even angry? Is he uncomfortable from inability to digest what he is taking? If he suffers pain, is it from colic? If he suffers from any upset during a previously smooth progress, what is the cause of it? Be assured, if you do not find out the cause, the upset will happen again.

Having made these preliminary observations let us grapple with our subject. Of all infant foods human breast milk is not only the best; it is incomparably the best. Especially is this so in the first few months. Though we have always acknowledged this, we have not always acted on it. We have not sufficiently realized its importance. Of late years and this is the most encouraging recent development in pædiatrics, the advocacy of breast-feeding, largely through the driving force of that fiery spirit Sir Truby King and the nurses trained in the institutions established through his efforts, has revived so greatly as to have produced a real effect in decreasing infantile mortality. And yet I am continually seeing infants who have been weaned early, often with disastrous results, for altogether insufficient or bad reasons or because the mother's milk "did not agree with the baby" in her own opinion or that of her friends, sometimes with the consent or even on the advice of her physician. Let me make my meaning plain by answering a few direct questions. Should a baby on the breast be weaned because he is passing curds in frequent loose motions, is fretful and unhappy and sleeps badly? Certainly not; unless suffering from some passing infection, he is being overfed; his feeds are too large or too frequent and need adjusting. Or because he is always crying with hunger, not gaining weight or losing it? Certainly not; the breast milk is insufficient in quantity, it must be increased or, if that cannot be done, some other food must be given at the end of each suckling to supply the want. Or because he is constantly troubled with wind, colic and vomiting? Again no. The trouble is in the baby, not in the milk. On artificial food he will probably be no better and may be much worse. Is there then no good cause for weaning from the baby's side? None that I know of. If any such exists, it must be extremely rare. Weaning may be necessary if the mother is suffering from tuberculosis or other chronic enfeebling disease, or if pregnancy supervenes. In the last instance it is not necessary to act hurriedly. Weaning should not be necessary in short, sharp illness; here only a temporary suspension of breast feeding is indicated. A baby should not be weaned because its mother has an abscess of the breast. Unless pus discharges into the nipple or milk-ducts, which seldom happens, the infant may take both breasts. Here I would protest against the custom of sacrificing a baby by sending the mother into hospital without her baby for some operation during the suckling period, the hospital should accept responsibility for infant as well as mother.

The induction of breast feeding is a question of good nursing. It is not always easy. Unfortunately many of our midwifery nurses in the past received no training in the care of the infant. In some of our nursing homes the only object appears to be to keep the baby from crying at whatever cost to his future welfare. He is turned out frequently wholly or partly bottle-fed, to take his chance of life, which is sometimes a poor one. Worse still, his cries are stilled by stuffing a dummy dipped in honey, malt, or other sweetmeat into his mouth. This practice, if persisted in, will cause dyspepsia even in a healthy breast-fed infant. I have known it to cause convulsions. So frequent has it become that in our clinic we find it necessary to ask: "What do you put on the baby's dummy?" For these evils, medical men must be held largely responsible. Too often they make no inquiry as to what treatment the baby is receiving.

The course of breast-feeding does not always run smoothly. Not infrequently we come across a fine, fat baby who is upsetting a whole household and its neighbours by yelling through the night. Perhaps it fortunately vomits, for this is a safeguard; if not it passes copious loose motions full of white particles. The remedy is simple. Feed it every four hours five times a day and not at night; restrict the length of its feeds; if necessary give it a tablespoonful or two of water before each feed. It is remarkable that usually the mother and her friends think this baby is not getting enough and proceed to give it artificial food or even to wean it altogether. Many a baby has been lost in this way.

The secretion of milk depends on two factors, internal and external. Of the former we know very little; the latter is very much under our control. It is the frequent stimulation of sucking at regular intervals that maintains the secretion. If the baby

stops sucking, the milk disappears. Unfortunately if the baby does not get all he wants by sucking, he may go on strike and refuse to help you. Especially will he do this, if he has obtained nourishment by the easier process of sucking a bottle. Here the only resource is the skilled nurse who can obtain milk from the breast by expression and so stimulate the flow. The expressed milk is spoon-fed to the infant while he learns to do his part. In this way we are constantly establishing satisfactory breast-feeding at our clinic and even restoring it after an abeyance of many weeks. Should you order an insufficiently nourished, breast-fed infant one or two supplementary artificial feeds daily, almost invariably the supply of breast milk lessens. You are in fact weaning him gradually. Your right course is to insist on his taking the breast regularly at every meal and, if necessary, to add a small complement of artificial food at the end of suckling to supply the deficiency.

The most difficult cases of breast-feeding are those in which the infant is troubled with persistent vomiting, colic and wind in the stomach. I am not alluding to the common habit of bringing up a teaspoonful or two after feeding. This should be called possetting, not vomiting; it is messy, but not harmful; indeed it may be considered a sign of health. Nor do I allude to the so-called hypertrophic pyloric stenosis with palpable tumour, which may need treatment by operation, but to a similar but more common affection of less severity with no apparent pyloric thickening. The stools are normal or those of starvation and there is no real dyspepsia. It is an unfortunate though common error which may lead to death, to regard the mother's milk as the cause of the trouble and to wean the baby. The real condition is one of gastric hypertonus and incoordination with pyloric spasm. These infants are of nervous temperament; all their muscles are generally overstrung and overactive; they sleep badly and they cry and stiffen themselves with much vigour. The mother herself is usually also of nervous temperament and over anxious. It is well to warn her at once that she has a difficult baby which will give much trouble, but with care will ultimately do well. Every effort should be made to continue breast-feeding. Hunger makes the babe worse. Sometimes, but not always, it does better on smaller feedings at more frequent intervals. Quiet handling is very necessary on account of the nervous unrest. Hot baths or packs may favour sleep. Wrapping up the limbs, as with the old-fashioned swaddling clothes, may be of help. Treatment by atropine to diminish vagus activity has been advocated; of this I have no experience. A daily stomach washout with warm water containing a teaspoonful of carbonate of soda to half a litre may be helpful. As long as the infant gains weight, however slowly, there is no need for anxiety and the symptoms will slowly disappear. In some cases there is much air swallowing during suckling. The infant's stomach becomes rapidly overdistended: he stops sucking and is restless and crying. Hunger and sleeplessness may increase, while the mother's milk diminishes. The same breast milk given with a bottle may be taken quietly, as may some modification of cow's milk, but the latter must be watched with care. It is the excited, restless, over-eager, hungry baby who swallows too much air. The same baby, when half-asleep, may take the breast quietly and easily. It is said that a tablespoonful or two of thick gruel before suckling stimulates the early contraction of the stomach on its contents and prevents overdistension with air. I have not so far tried this.

Sometimes a mother comes to you with a half-starved breast-fed baby and the request that her milk be analysed. She assures you that she has plenty of it, as it is constantly running away. Invariably you will find by a test feed, that is weighing the baby before and after suckling, that her milk is deficient in quality. Milk analyses are seldom necessary; to be of any value the sample must consist of the whole contents of the breast carefully emptied. Human milk varies much in composition, the fats from 3·5% to 5%, the proteins from under 1% to nearly 3% and yet within these limits appears to be equally wholesome. If Nature teaches us anything, it is that the exact percentage composition of breast milk is not of much consequence. We had a very striking illustration of this lately at our clinic. A wet nurse imported to supply milk for two premature infants was found to be supplying milk containing only 1·8% of fat and 4·2% of solids not fat (as against the normal 7% to 9%). We discarded her and tried a second who gave 2·8% of fat and 6·7% of solids not fat. Yet both these were healthy young women with healthy, well-nourished infants. But the premature babies, bottle-fed on rationed quantities of the milk of both women, suffered woefully and gave us an anxious time.

Strive as we may to keep all infants at the breast, there will always be many whom we have to feed artificially. Cow's milk, however modified, is never a perfect food; dried and condensed milks are still inferior, though they have their uses; proprietary milk-food mixtures I have discarded altogether. It is fortunate that most infants have a considerable margin of digestive power that enables them to thrive more or less on what is not their natural food. I am not one of those who hold that there is only one right way of feeding an infant on cow's milk, though I believe that some methods are better than others. On the other hand I am firmly convinced that better results will be obtained from an inferior food used intelligently than from a better food used without intelligence. It is good to calculate our quantities and percentages, but it is important to remember that we are dealing with variable factors and the baby is the most variable factor of all. I read lately of a young physician who was consulted about a baby's food. He asked its age and weight, took out a pad and pencil, figured out how many calories the baby needed, made this up by the necessary quantity of diluted milk and sugar, divided by five and ordered this to

be given every four hours. The mother was not impressed. She thought he might have looked at the baby, inquired about its stools and symptoms and so on. I agree with the mother. Someone has wittily observed "you cannot feed a baby with arithmetic," but without some arithmetic you will not know what you are giving him. If we used nothing but cow's milk we might calculate by quantity, but as we frequently add sugars and fats, we need some common measure and this is furnished by heat or energy unit, the calory. Cow's milk contains between 70 and 80 calories per hundred cubic centimetres (20 to 25 calories per fluid ounce), usually reckoned as 74, sugar 430, 16% cream 190 and so on. If some thorough-going dogmatist informs you that an infant, aged four months, needs exactly 110 calories per kilogram of body weight every twenty-four hours, I advise you not to believe him. A very good authority says that an active baby may need up to 40% more energy units than a phlegmatic baby. A thin baby loses more heat units per unit of weight than a fat baby. A previously starved baby needs more calories for growth than a well nourished babe of the same weight. But making all these allowances and carefully considering each individual case, you may find the 110 calory formula a useful general guide. Do not count your calories without being sure of your quantities, especially where solids are used. We are apt to order teaspoonfuls of sugar in a very loose way. There are only two sorts of teaspoonfuls that can be measured with any approach to accuracy—the flat teaspoonful from which the surplus is scraped off with a knife without packing and the heaped teaspoonful, but with most women a teaspoonful is neither of these, but anything between them. The other day I took up a teaspoonful and carefully weighed four flat teaspoonfuls of ordinary finely granulated white sugar 3·175, 2·980, 2·851 and 2·722 grammes (49, 46, 44 and 42 grains) respectively. Four heaped teaspoonfuls weighed 7·776, 7·387, 7·582 and 7·831 grammes (120, 114, 117 and 121 grains) respectively. I then called in separately two ladies of my household, both good cooks, and asked each of them to measure out an ordinary teaspoonful. The first weighed 5·637 grammes (87 grains), the second 7·193 grammes (111 grains)! I advise you to order flat teaspoonfuls and to see the spoon or to use one of the spoons issued by the manufacturers of dried milks.

It is well to have a clear idea of the percentage composition of the food you are using. Some years ago percentage feeding was carried out very elaborately in the United States. More recently Sir Truby King has strongly advocated a percentage modification of cow's milk by dilution and the addition of lactose and cream, which he calls "humanized milk." I have already pointed out that human milk varies greatly in its percentage of protein and fat. We can strike an average from our analyses, but few sucklings get average human milk and these do not get it at every meal. Waiving this point, which is of minor importance, we must point out that "humanized milk," despite its percentage composition and human milk are extremely different; the only point in which they agree is in their content of milk-sugar. In the former the casein predominates over the lact-albumin, lactoglobulin *et cetera* in the proportion of three-quarters to five-sixths, in the latter there is less casein than albumin. Furthermore the casein and albumin of cow's milk are not the same substances as those of human milk. In cow's milk the volatile fatty acids comprise about 10% of the fat, in human milk about 1·5%. The ash of cow's milk is three times that of human milk; the calcium is four times as much, the potassium three times. The term "humanized" for this modification of cow's milk was not wisely chosen. Unfortunately the bulk of mankind is more influenced by the labels that are put upon things, than by their real composition. They imagine that "humanized milk" is something very like human milk and very nearly if not quite as good food for babies. Do not think that I deny that this modified milk is a good food or that some babies do well on it. The practical difficulty of obtaining sufficiently sterile top milk or cream restricts its use in this State. Sir Truby King advises the substitution of an artificial emulsion of fats to supply their place. As to the digestibility of this I express no opinion; but to call anything "humanized milk" which has been modified by the addition of a substance consisting partly of peanut oil, is surely a strange use of language. It is agreed by most pædiatricians that high fat percentages, especially in hot weather, often disagree. I note with approval that advocates of this "humanized milk" resort to weaker mixtures in commencing feeding and endeavour to increase their strength gradually. In so doing they tacitly recognize that it is a food, like other modified milk foods which the baby has to be trained gradually to digest.

Probably like myself you have seen infants apparently doing extremely well on very different modifications of cow's milk, on dried milks and even on condensed milk and have seen other infants doing very badly on the same mixtures. There is no doubt that many infants have done well on plain undiluted cow's milk. It has been argued that dilution of cow's milk is a mistake, because this deprives the infant of albumin which is relatively deficient in even undiluted cow's milk and cannot be replaced by casein. If the infant can digest the casein I see no harm in giving it, for the fear of an excessive assimilation of protein appears to be purely theoretical and unsupported by clinical observation. But if he cannot, it would appear to be more rational to use whey-milk mixtures, as I have done myself in some cases. Do not let us forget that nutrition depends on assimilation, not on ingestion and that the failure to assimilate one ingredient of the food supplied may lead to the failure to assimilate any of it. Let us frankly recognize that the casein and fat of cow's milk are foreign foods which some infants may be trained to digest in high percentages, but which cannot safely be given in these percentages to all infants, that infants may do equally well on foods of very dif-

ferent percentage composition, that in every instance it is wise to begin with lower percentages than the supposed optimum and to work up to it gradually and that each infant presented to us is an individual problem which we must study carefully by the methods of trial and error. Experience will show that while with low protein relatively high carbohydrate and fat may be tolerated, with high protein the carbohydrate and fat must be kept low. Anyone who does not do this, is looking for trouble and will get it.

My own practice has been intermediate between these two methods. I have usually aimed at a dilution of two parts of milk to one of water, with the addition of small quantities of carbohydrates and only seldom do I add fats. After the sixth month I sometimes reduce the proportion of water and believe that most infants may be educated to digest undiluted milk at or before twelve months. Milk-sugar does not seem to have any advantage over cane-sugar, except in complementary feeding of infants at the breast, when sweetness in the artificial food should be avoided, lest the infant get too fond of it and refuse the breast. Of course we should expect *a priori* milk-sugar to be more digestible, but in practice the cheaper cane-sugar will be found almost always to agree equally well. The optimum quantity of milk is usually between 94 and 125 cubic centimetres per kilogram (1½ to 2 ounces per pound) weight *per diem*. A healthy infant doing well usually approaches the latter figure. Our milk supply in Queensland is unsafe unless boiled; only when the parents keep their own cow do I permit unboiled milk, for I have seen serious infections from its use. I have been very pleased to find that a good American authority who has used both raw and boiled cow's milk, finds the latter more digestible. To bring milk to the boil for a moment does not destroy its vitamins; no doubt vitamin *C* may be destroyed by prolonged boiling. Though I have seen many cases of infantile scurvy, I do not remember having seen one which I could trace to the use of boiled milk. In any case a little daily orange juice in the later months is a perfect safeguard. As an incidental result of the Queensland practice of boiling milk, all forms of tuberculosis are rare in children; they do occur, but the incidence of their occurrence is very low.

Many babies have great difficulty in digesting the curd of cow's milk. This is lessened by boiling and by dilution with water. The Americans have great faith in thin barley water (one tablespoonful of pearl barley boiled in half a litre of water for one hour or more and made up to the half-litre before straining); there is no doubt that the small quantity of contained starch is well tolerated by very young infants. Very seldom do I order a greater dilution than one part of water to one of milk. Dilutions of one in three if persisted in for more than a few days lead to semi-starvation. In very young infants or those of weak digestion I add citrate of soda 0·23 to 0·46 gramme to each hundred cubic centimetres (one or two grains to each ounce) of milk, but not more than 0·25 or 0·3 gramme (four or five grains) to each feed. In these also I find the addition of dextrimaltose (Mellin's Food) very often better tolerated than cane-sugar or milk-sugar. Usually I increase the proportion of milk to two-thirds, but should the infant prove intolerant of this, I would endeavour to make up the necessary calories by increasing the carbohydrate and cautiously adding a little fat. In every case one commences with a rather low dietary and increases it gradually to what one considers an optimum, feeling one's way carefully. When simpler methods fail I instruct the mother how to peptonize the milk. To 568 cubic centimetres (one pint) of milk are added 284 cubic centimetres (half a pint) of water, the mixture is brought to blood heat, one tubeful of Fairchild's "Zymine" powder added, kept at blood heat for thirty or more often forty-five minutes, then boiled. The mother will know when she has peptonized sufficiently by the slight bitter taste developed. Lesser degrees of peptonization are of little use. A little added dextrimaltose disguises the bitterness. This has proved extremely useful in a large number of infants and is not difficult to carry out, provided the mother is given clear directions in writing. It should indeed be an invariable rule to give written directions for every case of infant feeding specifying number, intervals and quantity of feeds in each twenty-four hours. When the infant is doing well, continue peptonizing until it has gained at least nine hundred grammes (two pounds) in weight; then attempt to lessen the peptonization gradually and finally to eliminate it.

These are not all the methods I have employed, but they are those which I have used most frequently and have found most trustworthy. I do not pretend that they are invariably successful and I am always ready to try new methods. At the present moment I am much attracted by the method of feeding with lactic acid milk recently described by Dr. Mayo, of Adelaide, and intend to give it a fair trial. Owing to our bad milk supply we have been forced in our clinic to depend largely on the use of dried milk. Both the well known brands are excellent nutrients and babies with strong digestions thrive on them perfectly. Unfortunately very many cannot tolerate them in full strength, higher dilutions have then to be used, perhaps supplemented by the addition of some carbohydrate. These infants do only moderately well, but if fed according to scale their digestion breaks down altogether. We see so many young infants who have become emaciated on these milk foods, that I do not advise their use in the first two months. Here condensed milk with its low protein and fat and high carbohydrate is often well tolerated.

Now and again you will meet with a young delicate infant who fails to thrive on any modification of cow's milk. Remember then, before it is too late, to make every effort to obtain for it its natural food. I know the difficulty of procuring a wet nurse, yet sometimes one can be procured and proves satisfactory. Of course, she must continue to suckle her own infant. Sometimes a relative or friend will come to the rescue. In a few cases the

maternity home of the Salvation Army has given me invaluable help. In this way you will save some lives which would otherwise be inevitably lost.

THE SIGNIFICANCE OF CERTAIN PATHOLOGICAL CHANGES IN THE CEREBRO-SPINAL FLUID.

By BASIL CORKILL, M.B., B.S. (Melbourne),
Biochemical Department, Alfred Hospital, Melbourne.

THE cerebro-spinal fluid is extremely sensitive to chemical changes in the blood plasma. Generally speaking the possibilities of the cerebro-spinal fluid as an aid to diagnosis are not fully realized. In this short paper it is proposed to deal chiefly with the variations found in the albuminous bodies, with special reference to Froin's syndrome.

MODE OF FORMATION OF THE CEREBRO-SPINAL FLUID.

The chorioid plexuses of the lateral, third and fourth ventricles play the chief rôle in the production of the cerebro-spinal fluid. The exact mechanism of production is at present only a purely speculative conception and need not concern us. The fluid formed in the lateral and third ventricles passes through the *iter* or Sylvius to the fourth ventricle, where it mixes with the fluid formed by the small *plexus quarti ventriculi*. From thence it escapes by the foramen of Magendie and the two lateral foramina of Luschka. Passing into the *cisterna magna*, it then diffuses downwards in the subarachnoid space surrounding the cord and upwards by the pontine and basal cisterns. Over the brain surface the cerebro-spinal fluid gains access to the large venous sinuses and lacunæ which constitute its chief means of communication with the blood stream.

FUNCTIONS OF THE CEREBRO-SPINAL FLUID.

The chief function of the fluid is to provide a water cushion for the brain and spinal cord. Such a cushion acts as a protection against sudden shocks. A second function is to regulate cranial capacity. With vascular congestion some fluid escapes in order to relieve the intracranial tension. In addition the cerebro-spinal fluid probably plays an important rôle in the metabolism of the brain, forming perhaps a medium for the transference of waste metabolites and the like.

THE NORMAL CEREBRO-SPINAL FLUID.

Macroscopically the normal fluid is perfectly clear and colourless. The fluid may have a slight opalescence or turbidity; this is generally due to an increase in the cellular elements and can be at once verified by microscopical examination. Slight, accidental blood contamination may be difficult to distinguish macroscopically from a lymphocytosis. Centrifugalization will at once distinguish, as in the former case a deposit of red cells will occur.

Enumeration of Cellular Content.

For the examination of the cellular content of the cerebro-spinal fluid the apparatus of choice is the Fuchs-Rosenthal counting chamber. The method recommended by Greenway is as follows: To 0·45 cubic centimetre of cerebro-spinal fluid in a test tube is added 0·05 cubic centimetre of a 0·5% solution toluidin blue. The tube is shaken and allowed to stand for fifteen minutes. After this time the fluid in the tube is stirred with a fine, clean glass rod and sufficient of the mixture placed on the counting chamber. The total number of cells is counted and the result divided by three gives the number per cubic millimetre.

The depth of chamber is 0·2 millimetre and diameter is four millimetres, so that the volume of fluid over the ruled area is $^{16}/_{5}$ cubic millimetre. As the fluid is diluted nine in ten, the total number of cells is $^{16}/_{5}$ multiplied by $^{9}/_{10}$ = $^{144}/_{50}$ or approximately $^{150}/_{3}$. Hence the total number of cells divided by three represents the number per cubic millimetre.

Chemical Constituents.

According to Mestrezat the following substances occur in the normal cerebro-spinal fluid:

Chlorides	0·70% to 0·76%
Urea	0·003% to 0·010%
Glucose	0·048% to 0·058%
Albumin	0·03%

In addition traces of bicarbonates, phosphates, sulphates, calcium and magnesium are found. Greenway states that lymphocytes not more than five per cubic millimetre are normally found.

Chlorides.

The estimation of the chlorides is extremely simple and yields valuable information. It is stated that a raised percentage is indicative of renal inadequacy. We have not found this investigation of great value in cases of only moderate severity. When the blood urea content is only slightly raised, we found that as a rule the chloride estimations yield normal figures. However in the presence of gross lesions we found it a very useful estimation. The instance of a patient, brought into hospital comatose or tending towards coma, is that in which the estimation is useful. The highest figure we encountered was a value 0·95% with a blood urea content of 250 milligrammes per hundred cubic centimetres.

Perhaps the most important application of this investigation is in cases of suspected meningitis. Low values are found in connexion with tuberculous meningitis. Mestrezat regards a chloride figure of 0·5% as being of equal pathological significance to that of the finding of the tubercle bacillus. In our limited experience we have come to the conclusion that, although not always present, a low chloride figure is pathognomonic of tuberculous meningitis. The following two cases may illustrate the conditions found in tuberculous meningitis:

C.W., *ætatis* twenty-two years, was admitted on August 8, 1925, complaining of severe frontal headaches, drowsiness and pains in his limbs. After a few days the patient's condition was clinically that of a typical tuberculous meningitis; on August 11, 1925, the cerebro-spinal fluid was examined and reported on as follows:

Total albumin, 0·3% (Normal 0·03%).
Euglobulin present.
Lymphocytes, 50 per cubic millimetre.
Chlorides, 0·5%.

The diagnosis was verified *post mortem*.

Case II. was one in which the diagnosis was clinically not so well defined.

F.M., *ætatis* fifteen years, was admitted on August 7, 1925, with the diagnosis of enteric fever. For seven days he had complained of more or less constant frontal headaches. His bowels were quite regular, but vague abdominal pain had been present.

He vomited once on the day before admission.

On examination the boy was seen to be very drowsy. Neurologically nothing abnormal was discovered. His spleen was not palpable and no rash was present. A diffuse abdominal tenderness was present. On August 7, 1925, a Widal test of the blood was made, but no reaction was obtained. On August 10 the patient was more drowsy and some slight neck rigidity was noticed. His cerebro-spinal fluid was examined and reported on as follows:

Euglobulin present.
Total albumin, 0·25%.
Lymphocytes, 40 per cubic millimetre.
Chlorides, 0·56%.

On these findings a diagnosis of tuberculous meningitis was made and verified *post mortem*. The abdominal pain was also accounted for by the presence of *tabes mesenterica*.

Estimation.—The following solutions are required:

(i.) A standard solution of silver nitrate (5·814 grammes to one litre).

(ii.) A saturated solution of potassium chromate.

Two cubic centimetres of cerebro-spinal fluid with eight to ten cubic centimetres of distilled water are placed in a small flask. To insure alkalinity a trace of calcium carbonate is added and then a few drops of potassium to serve as an indicator. The silver nitrate solution is then run in from the burette until the lemon yellow colour is changed to pink.

The calculation is based on the fact that each cubic centimetre of silver nitrate solution precipitates one part per thousand of chlorides. Thus if six cubic centimetres were used, the chlorides would be 0·6%.

Glucose.

As would be expected the glucose content is raised in diabetes. However, in this disease it is rarely estimated, the blood sugar content being more frequently determined. In all cases of meningeal inflammation, whether acute, subacute or chronic, the percentage of glucose is decreased. In the initial stages of tuberculous meningitis the glucose content may be increased. The cause of this is obscure, but according to Greenfield it may be due to pressure on the pituitary gland. For its investigation Maclean's blood sugar method may be used.

Recently attention has been directed to the sugar content in encephalitis. The present consensus of opinion seems to be that a high sugar content (0·06% or more) in an otherwise normal fluid is suggestive, but not pathognomonic of lethargic encephalitis.

Urea.

The estimation of urea is useful in cases of renal inadequacy particularly with reference to uræmia. Any reading above 0·05% with a normal albumin content is indicative of renal insufficiency. According to Mestrezat, a figure of 0·3% denotes a fatal prognosis.

The Albuminous Bodies.

The cerebro-spinal fluid normally contains a small amount of albumin (0·013% to 0·030%) consisting of both serum globulin and serum albumin. In all forms of meningeal inflammation the albuminous bodies are increased, chiefly in regard to the serum albumin. Thus, generally speaking, we find that with a total albumin increase the proportion between serum globulin and serum albumin is about 1:8. In syphilitic diseases of a subacute and chronic nature, we find that contrary to the above the globulin increase becomes more pronounced, so that the proportion of serum globulin to serum albumin becomes 1:2 or 1:3. Thus, if we were examining the cerebro-spinal fluid of a patient with chronic cerebro-spinal syphilis, we would expect to find a globulin reaction together with a total albumin figure of about 0·04%.

Occasionally, we have found in persons suffering from cerebro-spinal syphilis a globulin increase associated with a total albumin figure of 0·09% to 0·06%, that is the ratio of globulin to albumin would be about 1:4 to 1:5. However, these cases are generally exceptional to the figures quoted above and clinically are not difficult to differentiate from other forms of meningitis.

In all the more acute forms of meningitis, whether tuberculous, meningococcal or syphilitic, the albumin content rises to 0·2% or even 0·3%. Meningococcal meningitis especially gives very high figures. In these cases the ratio of globulin to serum albumin may be 1:12. In these more acute forms fibrinogen is present, so that a web coagulum forms, soon after the fluid is withdrawn. This coagulum is frequently seen in tuberculous meningitis. The onset and clinical signs help to differentiate tuberculous and meningococcal meningitis. In the tuberculous form excess of lymphocytes is found, sometimes up to 300 or more per cubic millimetre. A lymphocytic increase is also found in all forms of syphilitic meningitis. With meningococcal meningitis, we mainly get a polymorphonuclear leucocytosis and the organism may frequently be cultured from the cerebro-spinal fluid.

Froin's Loculation Syndrome.

Froin's loculation syndrome has shown itself to be of extreme importance in connexion with compression paraplegia and allied lesions. The following description is slightly modified from Greenfield's excellent account of the syndrome.

When for any reason the cerebro-spinal fluid is shut off partially or completely from access to the lower spinal theca, the fluid obtained by lumbar puncture is altered in certain definite ways.

The chief change is found in the albuminous elements which become greatly increased. In fact it is really this increase that constitutes the essential part of the syndrome. The albumin and globulin content approximates that of the blood serum. In its typical form Froin's syndrome is as follows:

1. The fluid when withdrawn has a yellow colour, varying from a definite yellow to a faint colour only visible when looked at down the long axis of the test tube.

2. It may coagulate *en masse* after withdrawal or in less pronounced cases only a fine web coagulum may occur.

3. The protein elements approximate the proportions which occur in blood plasma.

Pathology of the Syndrome.

A continuous circulation of fluid occurs within the spinal theca. Fluid in the lumbar region is being continually pumped from above by arterial and venous pulsation. Hence, if the ventricular fluid loses its communication with the lumbar, the latter must to a large extent become stagnant. Under such a condition there will be a constant admixture with lymph chiefly by way of the perineural lymphatics and soon the encysted fluid will have a protein content very similar to that of blood plasma.

There are two main groups of cases in which Froin's syndrome is encountered, acute meningeal affections, tuberculous, meningococcal or syphilitic and spinal neoplasms.

It is the second group that I propose to consider in the following discussion. Clinically it is easy to differentiate the two groups and there is no necessity to enter into a differential discussion.

Methods for Determining Protein Content of the Cerebro-Spinal Fluid.

Globulin.

In a Wassermann tube place 0·6 cubic centimetre of cerebro-spinal fluid and with a pipette run in 0·6 cubic centimetre of a saturated ammonium sulphate solution, so that it forms a separate layer below the cerebro-spinal fluid. An opalescent ring appearing within five minutes denotes a reaction.

Total Albumin Estimation.

To estimate the total albumin content it is necessary to have a series of standard dilutions of albumin ranging from 0·1% to 0·01%. The easiest method is to make a series of dilutions from normal human blood serum on which a Kjeldahl determination has first been made to ascertain the protein content. To make the standards take two cubic centimetres of the diluted serum in a test tube and add 0·3 cubic centimetre of 30% dilution of trichloracetic acid and boil. This completely precipitates the albumin. The test tube may then be sealed off and the percentage albumin content labelled on the outside. To carry out the test two cubic centimetres of the cerebro-spinal fluid are placed in a test tube, equal in diameter to the standard and 0·3 cubic centimetre of trichloracetic acid dilution is added. The mixture is boiled and the precipitate of the unknown compared with the standards.

Dr. Mackeddie has included the clinical history of a patient (H.W.) in his recent article to illustrate Froin's syndrome (see THE MEDICAL JOURNAL OF AUSTRALIA, May 22, 1926, page 577).

I would add that on April 27, 1925, a lumbar puncture was performed and it was found that the cerebro-spinal fluid yielded all the information characteristic of Froin's syndrome. The colour was a pronounced yellow; a web coagulum was present, as was fibrinogen present; the globulin response was pathological and the total albumin content represented 0·3%.

A lipiodol plate revealed a block between the twelfth dorsal and first lumbar vertebra. Coincident with the lipiodol injection the *cisterna magna* fluid was examined and was found to be quite normal.

This was Froin's syndrome in its most typical form, but if the mechanism of the syndrome be reconsidered, it will readily be understood that partial blocks may cause changes which though not constituting a typical syndrome, yet are fundamentally of the same type. Frequently with much lower albumin percentages and in the absence of any yellow coloration there may be a strong presumption that we are dealing with the loculation syndrome, provided that the number of cells and other constituents are normal.

The following is a case of an atypical Froin's syndrome. Fuller notes appear in Dr. Mackeddie's article:

Mrs. E. L. was admitted on March 2, 1925, with a diagnosis of spastic paraplegia. On March 31 a lumbar puncture was performed. The cellular content was found to be normal; there was an increase in the globulin content and the total albumin represented 0·85%.

The loculation syndrome was thought of and at a later date fluid was examined from the *cisterna magna* and found to be quite normal. A lipiodol plate revealed an obstruction at the level of the eighth thoracic vertebra.

From the lumbar puncture findings alone it would have been impossible to exclude say syphilitic meningitis, but the fact that the cisterna fluid was quite normal proved a loculation syndrome to be present.

Conclusions.

Examination of the cerebro-spinal fluid yields valuable information in meningeal lesions.

A lumbar puncture examination combined with that of the *cisterna magna* fluid is of paramount importance in cases of compression paraplegia. Such examinations indicate the presence of a loculation syndrome which in turn is an indication for a lipiodol localization.

Reports of Cases.

NOTES ON A CASE OF STRIÆ ATROPHICÆ.

By W. E. GEORGE, M.B., Ch.M. (Sydney),
Australian Institute of Tropical Medicine, Townsville.

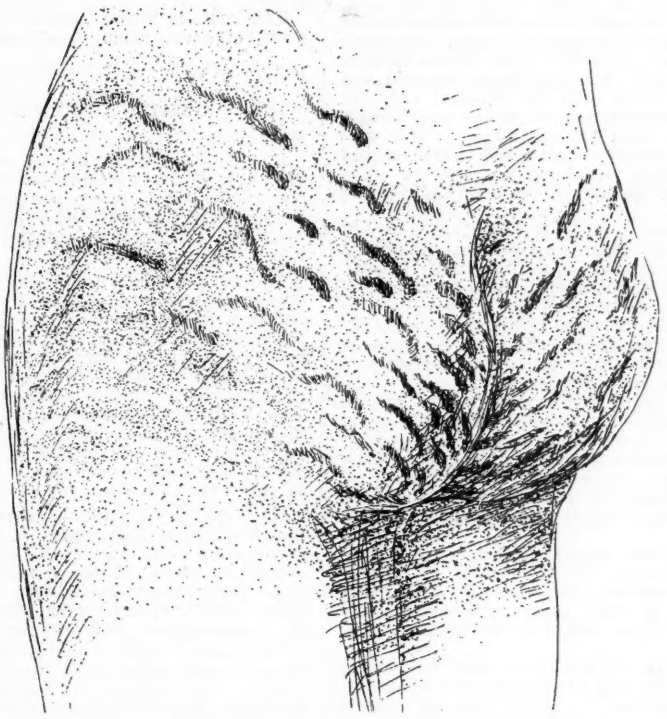
CASES of the relatively rare skin lesion known as *striæ atrophicæ* occur from time to time, the last recorded cases discoverable being noted by Hobbs (1923) who also gives a bibliography of the disease. The following case is a good example of the condition and is interesting both because no definite cause could be determined for the lesions and also because of their unusual localization.

The patient, M.C., is a girl, aged seventeen years. Six months before coming under observation she noticed "varicose veins" appearing over both buttocks and this has persisted up to the present. When the condition first appeared, the skin affected was very tender, especially when she sat down or stooped and there was continuous intense itching. The skin lesion has persisted since then, but there is now no pain, the only complaint being the presence of the lesions. The colour of the marks varies; it is more noticeable after exertion or when she is men-

struating. She has always been a delicate girl and has not played games like other children and she always feels tired and languid. Her appetite is fairly good, but she suffers with "bilious attacks" and has to be careful with her diet. She has no trouble with her bowels or urine and has had no previous illnesses except a "cyst" removed from a rib five years ago.

There is nothing of interest in the family history, she has ten half-brothers and sisters all healthy, but no full brothers or sisters.

On examination there were seen lines arranged symmetrically over the buttocks and resembling in appearance the *striæ gravidarum* appearing on the abdomen of a woman in the course of her first pregnancy. They had, however, a livid purplish colour, not the pinkish colour of *striæ gravidarum*. They were distributed in parallel rows perpendicular to the direction of the lines of maximum tension of the skin when the patient leaned forwards at the hips. The individual lines were irregular in outline and varied from 1·25 to five centimetres (a half to two inches) in length and were about three millimetres (one-eighth of an inch) wide. They commenced over the skin below the anterior superior iliac spines on each side and pursued an irregular course towards the folds of the buttocks. The arrangement was symmetrical and they were equally marked on each buttock. Over both hip regions the colour had already commenced to fade, leaving white linear scars similar to the typical *lineæ gravidarum*. Some slight tenderness still remained over the scars on deep pressure. On passing the finger transversely across the striæ, they could be be felt as definite elevations. There was no history of any distension of the skin nor of any violent bending or stretching movements. She has not been growing rapidly.



Figure, Showing *Striæ Atrophicæ*.

There was no evidence of disease of the heart or lungs, urine showed no abnormality; an X ray examination of the chest revealed no evidence of tuberculosis, the blood count, beyond a slight anæmia, was normal and the blood serum failed to react to the Wassermann test. Examination of the blood for microfilariæ yielded no result.

In the ten cases recorded by Hobbs five of the patients had definite tuberculous disease of the lungs, three had advanced disease of the heart of rheumatic origin, one had parenchymatous nephritis and one had a large liver abscess secondary to a small localized appendiceal abscess. The commonest sites in his case for striæ to appear were over the lower part of the thorax and above the patellæ. All his patients were between twelve and twenty-two years of age and in the absence of any evidence of rapid growth, distension by œdema or tumour he regards the condition as being due to an impaired nutrition of the skin caused by some constitutional disease and in these cases the main factor is that of posture, the striæ occurring at the positions of maximum tension. Apparently in the presence of severe malnutrition or some toxic condition about this age the elastic fibres in the deeper layers of the skin readily rupture.

Although no definite organic disease could be found in the patient, she was thin, anæmic and under developed and had lived all her life in the tropical coastal climate of Townsville. She has since been greatly benefited by a change inland where she gained five kilograms (eleven pounds) in weight in three weeks and now feels much stronger.

She is now (three months after first coming under observation) playing games and swimming and the striæ are gradually losing their purplish colour, becoming paler and no doubt will eventually disappear, leaving behind them, however, pearly white scars similar to those met with on the abdomen of a parous woman.

Acknowledgments.

I wish to express my thanks to Dr. W. Chapman, of Townsville, who referred the patient to the Australian Institute of Tropical Medicine, also to Miss Gladys Roberts for the drawing which demonstrates the condition far better than any written description.

Reference.

F. B. Hobbs: "Some Observations on Striæ Atrophicæ," *The Lancet*, September 1, 1923, page 452.

Reviews.

PROGRESS IN SURGERY.

THE year book on general surgery[1] for 1925 has reached us. It has been edited by the late Albert J. Oschner.

The book must not be misjudged. It is not a textbook, but a *résumé* of the most recent contributions to surgery. It is obviously a monument of revision and reading and the reader will be lost in admiration of the amount of work that must have been expended in its compilation.

The delver after recent knowledge will probably not search this volume in vain. Most of the later contributions have been reviewed. Apparently the book was finished after the appearance of the article by Gye and Barnard as no mention of their work is made in dealing with the causation of malignant neoplasms, but the student will find other theories to entertain him.

Gastric surgery is fully dealt with, but no didactic rules are laid down in dealing with the surgery of gastric and

[1] "The Practical Medicine Series, Comprising Eight Volumes on the Year's Progress in Medicine and Surgery": Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; Volume II.; General Surgery, Edited by Albert J. Ochsner, M.D., F.R.M.S., LL.D., F.A.C.S., F.R.C.S.Ir.(Hon.); 1925. Chicago: The Year Book Publishers. Crown 8vo., pp. 712, with illustrations. Price: $3·00 net.

duodenal ulcers. That is to say there is no decisive championing of one particular measure whether it be simple excision of the ulcer, excision with gastro-enterostomy or partial gastrectomy with or without gastro-enterostomy. Wisely no all-embracing dictum is attempted.

The effects of injecting alcohol into the Gasserian ganglion will prove of interest to many who will possibly find their own results conflicting with those here quoted.

The book may well be recommended as a work of reference and though the diction may jar at times, information may be obtained at that price.

The work is replete with many illustrations some of which are helpful.

ADVICE AND HELP FOR PARENTS.

A VOLUME that will be welcomed far and wide in the Commonwealth is Dr. Margaret Harper's "The Parent's Book."[1] The principles as its author states are not new. Rather they have borne the test of time and are confidently offered as sound teaching for the general public. Beginning with the expectant mother, the author gives simple rules of health with regard to diet, clothing, baths, exercise and rest and the dangers which call for medical aid, are enumerated. Minor troubles are discussed and simple remedies which may be used, are suggested. A few wise words on maternal impressions are added, which should save many mothers from unnecessary anxieties. The preparations required for the baby, his cot, clothes *et cetera* are next dealt with.

The chapter on natural feeding is important and full of the wisdom learned by practical experience, it should be a real factor in helping mothers to nurse their infants. Artificial feeding is then discussed and methods of modifying milk and the principles involved are explained. It is good to see that the misleading term "humanized" milk has been avoided. Standards for growth and food requirements follow and should lead to a more intelligent appreciation of the problems involved in infant feeding.

The chapter on the premature infant is also one of importance to the community. Many delicate babies are lost owing to ignorance in handling in the first hours or days of their life. Simple expedients available to most people are given. Then follow chapters on feeding of children up to two years, on minor ailments and on summer care of babies and finally some recipes and an index are added.

The book is well written, tersely expressed and full of the ripe fruit of experience. Dr. Harper is to be congratulated on her production. It is small. The whole effect is modest. This no doubt cheapens production and so makes it available to more, but it is worthy of a better setting.

It contains within its covers knowledge that the medical and nursing world need as well as mothers and when that knowledge becomes tradition, we may hope for results which will go far in populating this country with healthy children of our race.

A MANUAL ON OPHTHALMIC TREATMENT.

"OCULAR THERAPEUTICS" by Professor Franke, of Hamburg, has been translated into English by Dr. Loeb, of Chicago.[2] Any guide to successful treatment is of value. After so long an absence a work by a German professor is of unusual interest. One wonders how far research in Germany has been carried and what new therapeutic aids have been evolved.

This book is divided into two parts. The first deals with the various methods of treating ocular diseases. The section devoted to general treatment includes an excellent summary of the treatment of tuberculosis and syphilis. Mechanical and local medical treatment, serotherapy and electrotherapy and the value of cold and heat and the various rays are considered in the section on local treatment.

The second part of the book deals with the various ocular disorders. They are arranged on an anatomical basis and the appropriate therapeutic agents are discussed. Many of the drugs are little known to British oculists. Is this the explanation of our surprise at finding in only one place the phrase "treatment practically useless?" Will the treatment suggested for macular degeneration, disseminated chorioiditis and opacities in the vitreous and cornea justify the author's optimism?

This book contains a valuable contribution concerning protein therapy. The indications and directions for milk injections are thoroughly explained. The author considers that they are of preeminent value in gonococcal lesions and various forms of retinitis.

The book is written clearly and concisely and should prove of great value to all ophthalmologists.

RADIOLOGY AND THE JAWS.

DR. STANLEY COLYER'S book entitled "Chronic Infection of the Jaws" is a monograph dealing in particular with the radiographic interpretation of normal and morbid conditions of the teeth and adjacent tissues and in general with the relationship of focal infection of the teeth and jaws to systemic infection.[1]

The book opens with a concise account of the normal structures as seen in a dental radiogram and then passes on to a consideration of the characteristic appearances produced by inflammatory processes. Great stress is rightly laid on the importance of attempting to recognize the earliest changes produced by the infection, before the appearance of the periapical abscess and granuloma. The author also emphasizes the point that much valuable information as to the extent of infection and the patient's powers of resistance can be obtained by a minute study of the cancellous bony tissue of the jaws around and even at some distance from the dental roots.

No mention is made of other pathological conditions met with in a radiological study of the teeth, such as dentigerous cysts, impacted and unerupted teeth, foreign bodies *et cetera*. These conditions would obviously not come under the classification of chronic infections, but might have been enumerated and illustrated with reference to interpretation and differential diagnosis.

The author takes it as proved beyond dispute that a definite relationship exists between oral sepsis and general disease. He discusses the sources and predisposing causes of chronic jaw infection and then passes on to a consideration of the paths of systemic infection. From his experience which is evidently wide, he deduces the fact that in the vast majority of chronic invalids and especially in those suffering with general debility the original cause of the trouble could be traced to infected jaws, not necessarily manifesting gross infection.

The chapters on indications for and results of treatment and the causes of the failure of many general diseases to respond to dental treatment are well considered and many novel theories are introduced as an aid to prognosis.

The illustrations are excellent "negative reproductions" and, though an index is lacking, the book is small enough and arranged well enough for easy reference without it.

Altogether, the work can be thoroughly recommended to physicians and dentists alike as an authoritative and well presented monograph on the subject of oral sepsis.

[1] "The Parents' Book," by Margaret H. Harper, M.B., Ch.M.; 1926. Sydney: Angus and Robertson, Limited. Crown 8vo., pp. 89, with illustrations.

[2] "Ocular Therapeutics: A Manual for the Student and the Practitioner," by Doctor Ernst Franke, translated by Clarence Loeb, A.M., M.D.. 1925, St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 183. Price: $3·50.

[1] "Chronic Infection of the Jaws: A Short Radiological and Clinical Study," by Stanley Colyer, M.D. (London), M.R.C.P., D.M.R.E., with a Synopsis of a Series of Fifty Illustrative Cases by Reginald Curnock, L.R.C.P., M.R.C.S., L.D.S.; 1926. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 86, with illustrations. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 12, 1926.

A Medical Library.

THE written word is the only vehicle for the propagation of knowledge. The spoken word can be used to a limited extent. By its means individuals may gain information of a valuable nature, but it has the inherent defects of the absence of permanence, of the difficulty of applying critical analysis and of the fact that the details sought at a subsequent date are not available. Science could not progress without literature. Books, periodicals, pamphlets, reports and indices are the structures on which science depends. There are approximately two thousand medical periodicals published in the world today. The Library of the Surgeon-General's Office at Washington contains over a third of a million medical books and close on a half of a million pamphlets. Some conception can be gained of the magnitude of medical literature from the Index Catalogue of the Library of the Surgeon-General's Office. It was started in 1880 and approximately one volume was published each year. The tomes are massive, of foolscap size and nearly three inches thick. The letter Z was reached in 1895, when the fourteenth volume was published. The second series comprises twenty-one volumes and a third series is now well under way, five volumes having appeared. There are now forty-two ponderous books filled with the names of authors, titles of articles or books, places of publication and similar details. But the information does not represent the whole of medical literature because it takes something like fifteen years to complete the cycle arranged in alphabetical order. Very little of this store of knowledge is available in Australia. There are a dozen small, starved libraries. There is a considerable amount of reduplication, particularly of periodicals. No system has been evolved by means of which the available material could be accessible to all earnest students in the Commonwealth.

In the February issue of the *Long Island Medical Journal* there is a very valuable article by Mr. Charles Frankenberger, the Librarian of the Medical Society of the County of Kings. This library is said to be the fifth largest medical library in the United States of America and ranks in size with the Library of the Royal College of Surgeons of England. Mr. Frankenberger tells the story of the birth and growth of this great library. He gives a full account of its value to the medical world, not only in the State of New York. He describes the method of arrangement and of cataloguing and adds a general classification of the works contained in the library. Finally he gives an admirable lecture on the best method of using the Index-Catalogue of the Library of the Surgeon-General's Office and the *Index Medicus*, the two monumental keys to medical literature. He appeals for gifts of books, periodicals, pamphlets and reprints.

This wonderful library has cost the Medical Society of the County of Kings very little money. In the early days medical practitioners bequeathed their libraries to the Society; other libraries and collections were given by the living and several very valuable private libraries were purchased by private subscription and presented to the Society. Some of the periodicals are purchased; the proprietors of the majority send complimentary copies as an act of courtesy. Authors and publishers of medical books are invited to spare one copy for this collection without charge. In this way the library has increased from sixty volumes in 1845 to about one hundred thousand in 1926. This has been achieved by a medical society of modest membership.

There is a lesson to be learned from the history of this great medical library. The Branches of the British Medical Association in Australia have made a plucky but somewhat ineffectual attempt to build medical libraries. The Medical Schools of the Universities of Sydney, Melbourne and Adelaide have adopted the usual method with partial success. There are some medical publications in the public libraries and in the libraries of other scientific

societies. When this journal was established, the exchange list was very small and by no means satisfactory. By arrangement the libraries of the New South Wales and the Victorian Branches of the British Medical Association have received these exchange journals in equal distribution after the editorial department has finished with them. The collection has grown and is now a valuable albeit a somewhat small one. There are about one hundred and twenty periodicals. In a similar manner the books received for review are divided equally between the two Branches. The reason why the other Branches receive no periodicals or books from the journal is that in 1914 the two Branches named withdrew their medical periodicals and thus each lost a small number of exchange journals. It is obvious that the present method of creating medical libraries in Australia is destined to failure. The cost, if the library is to be maintained by purchase, is beyond the reach of any of our organizations. The only practical scheme would be to concentrate all effort on one great library for the Commonwealth, to appeal strongly to the proprietors of every medical periodical in the world and to the publishers of medical books for free copies and to invite authors of pamphlets, articles and reports to send one copy to the library. Relatively small annual contributions from the Branches of the British Medical Association in Australia and from individuals would suffice for the salaries of a librarian and two or three assistants. Appeals should be made to the wealthier members of the medical profession to establish a fund for the purchase of a home for the library. Members of the profession should be urged to make provision in their wills to bequeath their books and periodicals to the library. Duplicates could be used in exchange with other libraries for amplifying the collection. If a start were made now, a very valuable library would be in existence in ten years and its contents could be placed at the disposal of the profession throughout the Commonwealth by means of a lending section. The organization would have to be carefully managed. Success would depend on persistence and steady work.

Current Comment.

THE MECHANISM OF "INSULIN" ACTION.

It is assumed that the therapeutic preparation "Insulin" represents the active principle of the islets of Langerhans, named by Schäfer insulin. Although it is known that "Insulin" on injection causes a reduction in the sugar content of the blood, no accurate information is available concerning the physico-chemical action either of the pancreatic hormone or of the therapeutic extract on the tissues of the body and on the carbohydrate undergoing metabolism.

Dr. E. F. Mueller, Dr. H. J. Wiener and Dr. R. E. Wiener have recently published the results of an investigation into the mechanism of "Insulin" action.[1] They were led to undertake the investigation on considering the fact that the effect of "Insulin" on the body differs according to the method of its administration. If "Insulin" is given to rabbits intradermally in doses of one unit per kilogram of body weight, a greater fall in blood sugar is produced than when the same dose is given to the same animal by subcutaneous injection. When the "Insulin" is given subcutaneously, the blood sugar after one hour is found to be about 50% of its original value; after about four hours the value returns to normal. When the injection is given intradermally the blood sugar falls to a lower level at the end of the second hour than it does after subcutaneous injection and at the end of the sixth hour is still below the normal value.

Dr. Mueller and his collaborators carried out a series of observations on normal persons and on persons suffering from *diabetes mellitus* of varying degrees of severity. Necrosis of the skin has been reported by some observers after intradermal injections of "Insulin." Dr. Mueller and his fellow workers have not experienced this complication. In all their tests they used "Insulin" of U40 strength and they found that from five to twenty units could be given intradermally in from one to three wheals. When non-diabetic subjects were given an injection by both methods the difference in the lowering of the blood sugar was small and not uniform. The difference was greatest when small amounts of "Insulin" were used. The reduction in blood sugar was most pronounced at the end of the first hour. With small doses the greatest effect was produced by the subcutaneous method and with larger doses the intradermal injection produced a slightly larger fall. With smaller doses recovery to normal took longer after intradermal injection and with larger doses the subcutaneous injection had the more prolonged effect. The reaction of the blood sugar level to the two methods of injection was more pronounced in patients suffering from varying degrees of diabetes. The difference in the reaction was not found to be a measure of the clinical severity of the disease. It was found, however, that the rapidity of the return of the blood sugar to its original level ran parallel to the clinical severity of

[1] *Archives of Internal Medicine*, April 15, 1926.

the disease. It is impossible to describe in detail the findings as set out in the graphs dealing with each of the groups. It must suffice to state that in the milder forms the greatest reduction in blood sugar occurred at an earlier stage than in the more severe types. In the moderately severe forms the greatest difference in the blood sugar lowering effect of the two injections occurred at the second and fourth hours. This difference was seen at a later period than in the milder types of the disease. In severe forms of diabetes the reaction to "Insulin" injections was quite different from that seen in the milder forms. During the first hour after injection the intradermal administration produced only a slight fall and sometimes an increase in the blood sugar level. During the second hour the drop was slight and became pronounced only during the third and fourth hours. The subcutaneous injection caused a slightly greater drop in the blood sugar level at the end of the first hour, a more pronounced effect at the second hour and the level reached at the end of the fourth hour was still below that reached following an intradermal injection of similar dosage. In other words the graph was almost exactly opposite to that obtained in milder forms of diabetes. The blood sugar of six patients suffering from diabetes was estimated at intervals of twenty minutes and a composite curve representing the findings is given. This curve shows that at the end of twenty minutes there is a fall in blood sugar after intradermal injection and practically no alteration in the same time after subcutaneous injection. This point is one on which Dr. Mueller and his collaborators lay most stress. They hold that the reduction after intradermal injection takes place before an effective amount of the hormone can possibly be present in the blood stream. One reason for this view is that no such effect is noticed twenty minutes after subcutaneous injection. It is also stated that the rapid fall in blood sugar after intradermal injection cannot be due to quicker absorption and entry of the hormone into the circulation as in that case the effect would disappear sooner than the effect produced by subcutaneous injection. As a matter of fact exactly the opposite is found. If the increased response to intradermal injection were due to quicker resorption and the entrance of larger quantities of the hormone into the blood stream, increasing the dose should result in an even greater difference in the lowering of the blood sugar between the two modes of injection. Dr. Mueller and his collaborators point out that the reverse actually takes place. They conclude that "Insulin" injected intradermally acts immediately by way of an autonomic nerve pathway on the liver and intensifies its glycogenetic function. They sought experimental evidence of such a stimulus by blocking the nerve fibres with atropine. Rabbits were injected with ten milligrammes of atropine per kilogram of body weight simultaneously with "Insulin." The effect of the intradermal injection was much less in the animals which had received atropine than in those which had not, the difference on an average being 50%. The average effect of the subcutaneous and of the intravenous injections was the same in the animals which had received atropine and those which had not. The conclusion reached is that the nerve action of intradermally injected "Insulin" is decreased or eliminated by the atropine. Apparently then Dr. Mueller and his collaborators do not regard the nerve pathway as being called into play when the drug is given subcutaneously or intravenously.

The work of these observers is of importance and the conclusions reached by them are ingenious. It is well, however, that careful and searching criticism should be applied to their arguments and observations so that hypothesis be not allowed to overshadow fact. In the early part of their article Dr. Mueller and his fellow workers refer to microscopical changes found in the tissues after intradermal and subcutaneous injections. They do not discriminate between these varieties in their description. They found œdema in the area of injection and degenerative changes in the walls of the vessels. The walls were partially destroyed, thrombi were present and some of the thrombi completely closed the vessels, while others only partially blocked them. The deeper layers of the skin are well supplied with vessels and although absorption would in all probability be slower in this situation, no mention has been made of the increased pressure to which the "Insulin" in the intradermal wheal would be subjected as compared with the "Insulin" in the loose subcutaneous tissue. This may not be a large factor. Stress is laid on the statement that intradermal injection cannot result in quicker absorption as in that case the effect should disappear sooner and that the opposite is what happens. These observers found practically the same reduction of blood sugar in animals which were given intravenous injections as in those which were treated subcutaneously. In the latter return to the original level was not so rapid. In fact the graph for subcutaneous administration seems to stand about midway between those for intravenous and intradermal administration. It would appear that the question might well be asked as to whether some at any rate of the phenomena noted on intradermal injection are not due to the fact that absorption is necessarily more prolonged. Against this, of course, is the observation that in some instances return to the original blood sugar level was quicker in intradermal injections of larger doses of "Insulin" than in subcutaneous injections. In regard to the use of atropine for paralysing the supposed pathway it may be pointed that the giving of atropine is not the same as section of the sympathetic nerves concerned. Atropine induces considerable flushing of the skin. The conditions existing therein would then bear more resemblance to those found in the subcutaneous tissues and possibly would be partly responsible for the findings.

These few observations have not covered the whole of the interesting work of Dr. Mueller and his collaborators. Sufficient has been said, however, to indicate the lines along which they have been working. Future work must include investigation into the qualitative as opposed to the quantitative action of "Insulin."

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Phacoanaphylactic Endoophthalmitis.

REVIEWING the previous work, A. Rötth (*Archives of Ophthalmology*, March, 1926) records that animals can be sensitized by the injections of lens protein and that reinjections cause anaphylactic shock. The results of rupture of the lens capsule in sensitized animals are doubtful and vary with the experimenter. The reaction cannot be considered anaphylactic. Moreover it is not proven that an animal's own lens protein can cause hypersensitivity. Verhoeff and Lemoine made intradermal tests and obtained reactions in about 8% of cases. In the author's experiments he used fall of temperature as the anaphylactic symptom. He found that heterologous lens protein absorbed from the anterior chamber can cause hypersensitivity, but rupture of the capsule in sensitized animals caused no reaction. The skin reaction cannot be considered as local anaphylaxis. Cataract operations were performed on two patients who gave skin reactions and no inflammation followed.

The Slit-Lamp in the Diagnosis of Glioma and Pseudoglioma.

ILLUSTRATING the difficulty in deciding between glioma and pseudoglioma in some cases, Harrison Butler relates that an erroneous diagnosis was made in three cases submitted to an ophthalmological society (*Archives of Ophthalmology*, March, 1926). The slit-lamp should help in the diagnosis. A child of five with a doubtful white mass in the vitreous was thus examined. White cells were seen circulating in the anterior chamber, the retrolental space was full of cells. From the slit-lamp examination the condition was almost certainly inflammatory. Notwithstanding this, the eye was enucleated. No tumour was found. The author will welcome an opportunity of examining a true glioma by the slit-lamp to satisfy himself there are no cells in the anterior chamber or retrolental space.

Inflammatory Pseudo-Tumour of the Orbit.

F. A. WILLIAMSON-NOBLE (*British Journal of Ophthalmology*, February, 1926) defines inflammatory pseudotumour of the orbit as a condition which so closely resembles an orbital tumour as to be indistinguishable from it, giving rise to proptosis and limitation of movement. Birch-Hirschfeld divides them into three groups. In the first of these are those cases in which recovery occurs spontaneously or with the administration of potassium iodide. In the second group are those in which an operation is performed and in which no tumour is found and the third group includes those in which the macroscopical appearances of the orbital contents suggest a tumour, but the microscopical appearances prove the lesion to be inflammatory. Three cases are described. The first patient was a man of twenty-five with proptosis of the right eye. Treatment with potassium iodide and mercurial inunctions failing to cure the condition, exenteration was performed. The orbital mass proved to be a gumma. The second patient was a middle-aged man with both eyes prominent and definite proptosis of the left eye associated with diplopia. His trouble followed influenza. A corneal ulcer developed, the eye was removed and the orbit exenterated. Examination of the mass revealed lymphocytic proliferation due to inflammatory change associated with the appearance of a lymphoma, the one to a certain extent merging into the other. The third case is an example of a small mass of young connective tissue forming outside the sclera as a result of irritation induced by the presence of cholesterin crystals.

Cerebral Aneurysm Causing Ocular Symptoms.

J. A. CONWAY (*British Journal of Ophthalmology*, February, 1926) reminds his readers that cerebral aneurysm is of frequent occurrence. Osler found twelve cases in eight hundred autopsies and Newton Pitt found nineteen in nine thousand inspections. They may cause no clinical symptoms or they may rupture and cause death. Q.D., aged twenty-three, was regarded as suffering from pituitary tumour with great alleviation of symptoms. He suffered from attacks of pain in the head for eight months. The right pupil was larger than the left and both discs were pale. Vision was extremely bad; he had bitemporal hemianopsia. Later he recovered from his blindness and resumed work. A few months later he died suddenly. At the *post mortem* examination there was free blood under the *dura mater* and the *sella turcica* was filled by an aneurysm. A second patient, a woman of forty, suffered for two years with pain in the back of the neck, headache, dizziness and defective vision. Shortly after admission to hospital she became stuporous. There was right facial palsy with paresis of the left arm and leg, the right pupil was larger than the left and she had double optic neuritis. A diagnosis of cerebellar tumour was made. The autopsy revealed an aneurysm of the left vertebral artery invading the cerebellum chiefly. In many cases there is congenital weakness of the vessel walls.

Vascular Lesions of the Retina in Young Persons.

VASCULAR lesions in late life have received much attention; H. Friedenwald has made a study of vascular changes in young subjects (*Archives of Ophthalmology*, March, 1926). In the first group he includes cases of recurrent retinal and vitreous hæmorrhages. He has recorded in a former paper definite changes in the calibre of the retinal vessels and especially of the veins, constrictions, dilatations, the formation of varices and of newly formed vessels projecting into the vitreous. Other observers have reported similar findings. Jackson and Finnoff have accepted completely the tuberculous nature of these changes and no other satisfactory explanation has been proposed. *Retinitis proliferans* is a common sequence. In the second group retinal disease with massive exudation and vascular anomalies are described, tortuous and engorged veins, aneurysmal dilatations and sclerosis of the vessels and perivasculitis. Coates ascribes the condition to vascular disease and hæmorrhage into the external layers of the retina and their subsequent organization. In the third group acute circumscribed exudative chorio-retinitis, vascular disease, is sometimes observed. The author has reported a perivasculitis, showing itself in numerous white spots arranged along the sides or upon the surface of the vessels. This condition may be associated with hæmorrhage into the retina and vitreous. In some conditions the vascular disease is the cause and in others the result of the retinal disease. The vascular changes in some cases may be congenital.

Lens Antigen Treatment of Cataract.

A. E. DAVIS reports results of treatment of a further series of fifty-nine patients and one hundred and nine cataracts with lens antigen (*Archives of Ophthalmology*, March, 1925). The first experiments were concerned with the injection of antilens serum prepared by immunizing rabbits and sheep against lenses or cataract of other animals. Later lens antigen was injected directly into patients. A sensitization test to the reaction of lens protein should first be given. The first therapeutic dose should be 0·5 cubic centimetre increasing by 0·5 or one cubic centimetre every other day until a maximum of eight to ten cubic centimetres is used. The dose should then be decreased for about ten days, when it can be gradually increased to toleration. Fifty doses constitute a course of treatment extending over four months. It may be advisable to give the injection every day. The results reported by the author are as follows: The progress of cataracts was arrested and vision was improved in eighty-six eyes, 65·64%; progress was arrested with improvement in vision in thirty-six eyes, 27·48%; in nine eyes the cataracts advanced. It is too early to speak of the durability of results.

LARYNGOLOGY AND OTOLOGY.

Septic Thrombosis of the Cavernous Sinus Following Furuncle of the Right Nasal Vestibule.

A. LOGAN TURNER AND F. ESMOND REYNOLDS (*Proceedings of the Royal Society of Medicine*, November, 1925) report a case of septic thrombosis of the cavernous sinus in a male patient, aged thirty-three years. The patient

suffered from a furuncle on the inner aspect of the right *ala nasi*. After rupture of the boil symptoms and signs of cavernous sinus thrombosis and of leptomeningitis occurred. After seven days the patient died. At autopsy the nasal cavities and accessory sinuses were found to be normal. The furuncle was regarded as the primary source of the infection. Microscopical examination revealed septic thrombosis of the cavernous and circular blood sinuses with retrograde thrombosis of the ophthalmic veins, the spheno-parietal sinus, the meningeal veins and the veins of the diploe in the walls of the sphenoidal air sinuses. James Dundas-Grant in discussing the case asked why so serious a result should have followed a furuncle. He mentioned three possibilities in connexion with it. The first was some distribution of veins peculiarly favourable to the extension of the suppuration. The second possibility was a special virulence of streptococci and the third was a specially low resistance on the part of the patient. He reminded the audience that Cheatle had recently shown that the mastoid emissary vein was sometimes of very large size. Cheatle had told him of a case in which a boil on the neck had led to thrombosis of the mastoid emissary vein, cavernous sinus thrombosis and death.

The Mastoid Emissary Vein.

ARTHUR H. CHEATLE (*Journal of Laryngology and Otology*, October, 1925) points out that the mastoid emissary vein is the vestigial remains of the primitive jugular vein. It is usually single and small, but is subject to variation in every particular. Its course is always in the substance of the bone, that is its canal is never surrounded by cells. It originates on the outer edge of the lateral sinus groove just below the bend and courses in an upward and backward direction. It opens on the surface just behind the upper posterior edge of the base of the mastoid process on a level with the roof of the bony auditory meatus and drains into the occipital or posterior auricular vein so as to form part of the external jugular vein. The vein may be opened by operative procedures in the vicinity, the bleeding, however, being easily controlled. The vein may be infected from the lateral sinus and an œdematous swelling or abscess may form at its exit, forming "Griesinger's sign" of lateral sinus thrombosis. The vein may be separately and directly the seat of septic thrombosis without implication of the lateral sinus.

Ionic Medication in Chronic Middle Ear Suppuration.

NORA LISTER (*Journal of Laryngology and Otology*, February, 1926) gives the results of treatment by ionic medication of thirty-eight patients suffering from chronic suppuration of the middle ear and of five patients with persistent aural discharge following radical mastoid operations. These patients were treated at the Royal Infirmary, Edinburgh. The technique is fully described. It follows that of Friel who has claimed 49·6% of cures by this method. The author has summarized her results and fully analysed the same as regards the causation and anatomical site of the lesions. In the first group forty-six ears were treated and of these thirteen became dry, three were stated by letter to be dry, five showed improvement, three were temporarily improved and twenty showed no improvement. In the second group (of five patients) six ears were treated, none was dry, one was improved, one was stated by letter to have improved, one was temporarily improved, three showed no improvement. These results indicate 25·5% cures and as the author points out, are not as brilliant as those reported by Friel and some others.

The Differential Diagnosis of Lateral Sinus Infection.

GEORGE L. TOBEY, JUNIOR (*Boston Medical and Surgical Journal*, January 24, 1926) discusses the diagnosis of lateral sinus infection. This lesion is always secondary to middle ear or mastoid suppuration. Symptoms of complications of a suppurative middle ear condition having arisen, the first step is to decide by careful examination the site of the complication. This may be intracranial or extracranial. The author enumerates many extracranial lesions which may complicate ear sepsis. The three intracranial complications are brain abscess, meningitis and sinus infection. Of these cerebellar abscess is usually associated with lateral sinus infection, while cerebral abscess and meningitis usually give characteristic signs. It must, however, be remembered that a lateral sinus infection will at the outset sometimes give signs of meningitis (meningismus). Lateral sinus infection varies greatly in its clinical signs and the author enumerates several types he has noted. In order to assist diagnosis, in conjunction with Dr. James B. Ayer he has investigated the dynamics of cerebro-spinal fluid and has evolved a new test, using a manometer in conjunction with lumbar puncture. In a normal person compression of both internal jugular veins causes a definite increase in the manometric pressure of the cerebro-spinal fluid. Previous experiments have shown that absence of rise in pressure on venous compression (positive result to the Queckenstedt test) indicates a subarachnoid block or a tumour in cerebellar fossa. Adapting this method for diagnosis of lateral sinus thrombosis, the author has evolved the following technique. Lumbar puncture is performed with the patient in the lateral position and the fluid is run into a glass manometer of two millimetres bore. Pressure on the internal jugular vein is then made, one side at a time and both together, manometer readings being taken on each occasion. In a typical lateral sinus infection compression of the normal side gives a rapid rise in manometric pressure, two to three times greater than normal. Compression of the vein on the diseased side gives no rise or a very slight rise, the result varying according as the sinus is obliterated or the seat of a mural thrombus. Eighty-four of these examinations yielded errors that may occur as follows. Patients whose cerebro-spinal fluid is under low tension, manifest no rise on compression of the vein. To remedy this twenty cubic centimetres of physiological saline solution are injected. The technique must be correct and gently used as coughing, grunting or movement of the patient will cause false reactions. Minor differences may occur normally on one side of a patient. The dangers are those attendant upon practising lumbar puncture on patients with intracranial lesions, but in the author's eighty-four cases no ill-effects were noted. The evidence adduced was found reliable and helpful in determining diagnosis.

Tracheal Tumour.

JAMES ADAM (*Journal of Laryngology and Otology*, March, 1926) reports four cases of tracheal tumour encountered in his practice. The first patient was a woman, aged forty years, who had a soft epithelioma near the right bronchus and who died six months later from lung metastasis, although operation and radium applications had been carried out. The second was a girl of eighteen who died eight months after treatment by radium and Erlangen irradiation. The tumour was situated above the right bronchus and although no autopsy was obtained, the reaction of the growth to treatment suggested it to be a sarcoma. The third was a man, aged thirty-four years, who was first seen when moribund. The growth was a fibroma situated anteriorly in the subglottic area. Treatment after an immediate tracheotomy was at first irradiation and later removal by crico-fissure. This patient died of pneumonia four weeks after operation. The author was of the opinion that preirradiation had complicated the operation. The fourth patient was a woman, aged thirty-seven years, in whom an endothelioma situated anteriorly at the fourth ring of the trachea was found *post mortem* after sudden death. Commenting on these cases, the author considers that symptoms of tracheal tumour are often mistaken for asthma, as was done in two of the reported cases or for phthisis. The combination of cough, occasional dyspnœa and hæmoptysis, with apparently good health and absence of thoracic signs suggests tracheal tumour and indicates endoscopic examination. X ray treatment before operation is not beneficial. Further, the risks of cartilage necrosis which follows X ray therapy are so great that the author questions the advisability of its use, when surgical treatment is possible.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Medical Society Hall, Albert Street, East Melbourne, on April 7, 1926, DR. H. DOUGLAS STEPHENS, the President, in the chair.

Workers' Compensation.

MR. R. G. MENZIES, Barrister-at-Law, addressed the meeting on medico-legal aspects of workers' compensation.

He pointed out that the condition of compensation was "personal injury by accident arising out of and in the course of the employment." The important words for present consideration were "personal injury by accident."

Mr. Menzies proceeded to discuss the definition of "accident." The view originally taken by the courts was that an accident was something fortuitous or abnormal, excluding things within the range of ordinary incidental risk.

A series of House of Lords decisions (1903-1919) had altered this conception and had established a very wide meaning for the term accident. That wide meaning might be illustrated by certain propositions which were to be deduced from decided cases.

(i.) "If a workman in the reasonable performance of his duties sustained a physiological injury as the result of the work he was engaged in . . . that was accidental injury in the sense of the statute."

This proposition was subject to later limiting propositions.

(ii.) The injury must be unintentional from the workman's point of view. Intentional injury inflicted by a third person might still be accident. Mr. Menzies cited cases in which a cashier was murdered, and in which a school teacher met his death as the result of a criminal conspiracy among students.

(iii.) In many cases the injury could be directly attributed to definite facts falling within the popular notion of accident. If examples were needed, they were provided by fractures, septic poisoning through a break in the skin *et cetera*.

(iv.) In many others the injury could not be directly attributed to definite objective facts and yet in result conformed to the idea of accident. Rupture of the aorta induced by over-exertion, cerebral hæmorrhage ensuing in a fireman who drank too much water while in the stokehold, nervous shock caused by witnessing an accident, had been held to fall within this group.

(v.) Accident, however, still connoted circumstances involving the unusual or unexpected. There was thus no accident where a seaman suffered illness by reason of the general exposure to rough weather incidental to his work.

(vi.) Something which was not an "accident arising out of and in the course of the employment" might have consequences or results which did come within the definition. A workman who suffered from fits, might have a seizure while at work. So far the statute did not apply. In consequence of the attack he fell and fractured his skull. It had been held that in such an event the statute did apply.

(vii.) The preceding propositions plainly excluded many diseases, notably industrial diseases. Hence amending legislation had made special provision for compensation for industrial or occupational diseases. These were set out in the fifth schedule of the *Workers' Compensation Act* and included anthrax, lead poisoning, mercury poisoning *et cetera*. As a rule these diseases did not fall within the notion of "accident," being of gradual onset and not associated with an event capable of being fixed.

(viii.) Although industrial and idiopathic diseases were excluded from the normal definition of "accident," diseases were included if contracted in or arising out of accidental circumstances, for example blood poisoning through handling artificial manure such as bone dust, erysipelas following injury.

(ix.) In all such cases the date and circumstances of onset must be capable of being fixed with reasonable accuracy, for accident still involved an event of which due notice under the Act might be given.

(x.) Diseases were also included if aggravated or accelerated by accident. It was sufficient if the accident was one of the contributing causes of the disease.

Examples were to be found in the case of a workman the subject of aneurysm of the aorta, who tightened a nut with a jerk and precipitated rupture of the aneurysm or in cases of crushed finger, septic infection, amputation, the sepsis aggravated by syphilis contracted between the crushing and the amputation.

The particular condition, of course, fell outside the statute if quite independent of the accident.

MR. L. I. SKELTON, of the Mercantile Mutual Fire, Accident and Marine Insurance Company, Limited, read a paper entitled: "Workers' Compensation Act: The Medico-Legal Aspect as Applied to Insurance Companies" (see page 659).

DR. JOHN KENNEDY read a paper entitled: "Some Medical Aspects of the Workers' Compensation Act" (see page 661).

DR. F. L. DAVIES said that he was not in agreement with the suggestion made by Mr. Skelton that medical certificates furnished to claimants for compensation under the *Workers' Compensation Act* should be framed in technical language. The patient who was paying for the certificate, was surely entitled to know the nature of his injury or disability.

He thought that the line which figured in accident insurance medical certificate forms requesting the certifying practitioner to state how the accident occurred might well be omitted. The patient or actual witnesses of the accident alone could describe the circumstances.

DR. WILLIAM OSTERMEYER said that the medical practitioner was often placed in a difficult position in answering the question: "How long would the disability last?" He was in the habit of stating "at least" a certain period. He had gained the impression that the insurance companies acted on the idea that if the claim were settled out of hand, the patient went back to work. Long experience had shown him that such was seldom the case. In his opinion compensation should be assessed at the time the worker resumed his employment.

MR. SKELTON in reply referred first to the point relating to diminished earning capacity raised by Dr. Kennedy. He pointed out that the English and Victorian *Workers' Compensation Acts* were similar in the sections dealing with diminished earning capacity and read the section of the Victorian *Act*.

Under the Fourth Schedule of the *Workers' Compensation Act* in Victoria specific percentages of the sum payable as for total incapacity were paid for certain injuries. If an injury (other than those mentioned in the Fourth Schedule) caused a permanent disability, even though it preclude the worker resuming his former avocation, as he read the *Act* such disability was not necessarily a factor in determining compensation. The individual's earning capacity in other directions might not be at all impaired. Notice was taken of permanent disability only when it in fact resulted in diminished earning capacity.

His remarks on the subject of medical certificates were intended as suggestions only. If medical men held different views, he was quite prepared to accede. In his opinion, however, any statement in a certificate likely to render a workman a burden on the community as the result of his knowledge of such a statement should not be put into the hands of the patient.

In reply to Dr. Ostermeyer, Mr. Skelton said that insurance companies settled only a few claims on the basis of the doctor's estimate of the period of incapacity. The companies' object in asking for an estimate of the period of incapacity was chiefly to gain assistance in gauging their outstanding liabilities. They also wished to be in a position to detect malingering. There was no desire on the part of insurance companies to penalize the worker in requesting this information and he hoped certifying practitioners would continue to supply it.

The request to state how the accident occurred, referred to by Dr. Davies, appeared on certificate forms relating to personal accident insurance but not on those in connexion with the *Workers' Compensation Act.*

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Laboratory of Pathology, Bacteriology and Biological Chemistry, Adelaide Hospital, on April 29, 1926. The meeting took the form of a series of demonstrations of laboratory methods of investigation and of pathological specimens.

Laboratory Aids to Diagnosis.

DR. L. B. BULL, Director of the Laboratory, said that it afforded him great pleasure to welcome the members at the laboratory that evening. They were attempting to interest members in laboratory methods, but more particularly those procedures which were used as laboratory aids to diagnosis in modern medicine. Professor Cleland had some pathological material which he would demonstrate. Dr. Lewis would briefly outline the methods adopted in testing renal efficiency and they would demonstrate the skin test used as an aid in the diagnosis of hydatid disease. Afterwards members would be asked to adjourn to the main laboratory where various tests would be shown. Before proceeding, however, he had a few remarks to make which might be of some interest and possibly of some help to the general practitioner particularly.

All new procedures in medicine were taken up with great enthusiasm by many workers. This was particularly true with regard to laboratory aids to diagnosis. This enthusiasm diminished and had a habit of disappearing entirely after clinical application had been thoroughly tried. For the most part too much was expected from laboratory aids to diagnosis. By this he did not mean that they should be used less, but that they should be used with greater judgement. He who obtained the greatest aid from these tests, was the one who used them with the greatest judgement. They all desired a test which would give 100% positive results and which would always be specific. Most of the laboratory tests fell far short of this. Many laboratory tests were far from being specific, but they might be very valuable to the clinician when he knew their limitations. Many of these tests merely revealed a state or condition of the body or body fluids and the determination of the presence or absence of a condition, although it would not be specific for any one disease, might be just as useful as the determination by clinical methods whether a state or condition, such as fever, existed in a patient. The clinician knew when he made a determination of the temperature of a patient just how much help he had obtained towards making a diagnosis. That determination might be a crucial piece of evidence, but it was only a piece of evidence. So many laboratory aids to diagnosis supplied nothing more than a piece of evidence, whilst a few might often supply the only evidence available and might be absolutely specific for one special disease.

There was a tendency to neglect many laboratory tests because they were not specific for one disease, but he believed that many of these tests would be used just as commonly as the clinical thermometer when they realized just how much information might be obtained from them.

There was a small and cheap book which some might find useful. It was "The Link Between the Practitioner and the Laboratory," by C. Fletcher and H. Maclean. It gave an alphabetical list of diseases against which were mentioned the laboratory tests which might be performed as an aid to diagnosis and the earliest time at which a report on the results of the examinations could be expected. Methods of collecting various specimens were given, but these might have to be modified somewhat according to the methods used by the pathologist consulted. Before collecting a specimen it was advisable to ascertain from the pathologist the manner in which he preferred such a specimen to be collected.

As he had been engaged in laboratory work in Adelaide for some years and had received specimens for examination from practitioners in many parts of the State, he might be permitted to mention certain common errors made in the collection of specimens for examination. Before going on to this, however, he would like to say that he believed that too little use was made of blood culture methods in the diagnosis of typhoid fever and other infective processes. One of the drawbacks to this procedure was the difficulty experienced by so many of obtaining specimens free from outside contamination. The asepsis which was adequate in surgical procedures, was often inadequate when applied to bacteriological procedures. They had found that boiling the apparatus in water was insufficient to obtain absolute sterility. The method adopted in their laboratory was as follows. A pad soaked with iodine was placed on the arm at the bend of the elbow and left there for ten minutes. This appeared to be more effective than cleaning up the skin by the usual methods and smearing some iodine on the surface. The blood was collected from the vein directly into a sterile syringe, five cubic centimetres being usually taken. This was immediately transferred into the same quantity of 1% citrated saline solution which had been sterilized under pressure. This was transferred in the laboratory to one hundred cubic centimetres of broth medium. The syringe used in collecting the blood was sterilized by placing the thoroughly dry instrument in pure chloroform for ten minutes or longer. The barrel, piston and needle were kept separate and when removed from the chloroform this was allowed to evaporate and it was necessary to get rid of it thoroughly. The dry syringe was then assembled and washed out with saline solution that had been sterilized under pressure. If the syringe was not moistened with saline solution, the blood would be drawn between the piston and barrel, making it difficult to work the piston smoothly and if clotting should occur great difficulty would be experienced in separating the parts. They had repeatedly experienced contaminations with spore-forming bacteria when saline solution as usually prepared in the wards was used for washing out syringes. With a little care it was an easy matter to obtain specimens of blood free from outside contamination and the general practitioner need not call in a pathologist to collect such specimens for him, although the pathologist would always prefer to collect the specimen himself.

The next matter he would like to touch on concerned the diagnosis of gonorrhœa. They commonly received smears from the vagina to be examined for gonococci and while, on occasion, these might be satisfactory, for the most part it was waste of time examining them. If smears were made from the urethra and cervix, satisfactory results would be obtained. The pus should be collected and immediately spread in a thin film on a glass slide. It was useless collecting the specimen on a sterile throat swab and sending that for examination. Commonly they received urethral discharge squeezed between two glass slides which had become stuck together as the discharge dried. Some very horrible specimens made in this way had been received and he would like to impress upon his audience that a film of the pus should be made immediately after collection and allowed to dry in the air. They always made their films as they would a blood film by using the edge of another slide and this was most satisfactory.

The examination of throat swabs for the detection of the diphtheria bacillus did not always give satisfaction and he took the opportunity of expressing the opinion that the diagnosis of diphtheria should not rest entirely on the bacteriological examination. The clinician should not wait for the result of the bacteriological examination before deciding upon the treatment. The examination was of very great value, but it must be recognized that the organism would occasionally not be found in undoubted cases of diphtheria.

With regard to laboratory tests as an aid in the diagnosis of typhoid fever too much was expected of the Widal test and blood culture was not used frequently enough. With blood culture a positive result would frequently be obtained very early in the disease, while the Widal test would usually not give a positive result until the eighteenth or even up to the twenty-first day. They had observed in

several cases that a positive result to the Widal test had not been obtained until convalescence. Blood for the Widal test should preferably be collected by the venepuncture, one to two cubic centimetres being collected into a hypodermic syringe and transferred to a dry, sterile test tube or small bottle. The collection of specimens of blood in small capillary tubes allowed only the microscopical test to be done and this did not give satisfactory results compared with the macroscopical test.

He would like to impress upon practitioners the value of possessing a hæmoglobinometer. Most of the best types were unfortunately rather expensive. For bedside work one giving a direct reading, such as the Dare, was the most satisfactory, but even the cheap and simple Tallquist scale would give a fair estimation of the hæmoglobin present. Such a preliminary examination would often save a lot of time and inconvenience as in his experience many people with approximately normal hæmoglobin values were suspected of being anæmic and were sent to the laboratory for complete blood examinations. For the purpose of estimating the cellular content of the blood the proper graduated pipettes must be used and the blood diluted with the appropriate fluids. It was useless sending clotted blood to the laboratory for this purpose, but they had experienced such a thing on more than one occasion. The examination of a blood film would often give very useful information. The film should be thinly spread on the slide by using the edge of another slide and should be allowed to dry in the air. On no account should the film be subjected to heating in a flame.

In the diagnosis of hydatid disease there were several tests which had been found to be useful. The complement fixation test was of value, but in their experience it yielded positive results in only approximately 50% of patients proved to have hydatid disease at operation or *post mortem*. It was more likely to yield a reaction when there was some degeneration in the cyst wall. For this test the blood was collected by venepuncture, five cubic centimetres being placed in a dry sterile test tube or bottle and allowed to clot. In sending blood from a distance to the laboratory it was advisable to decant the serum from the clot and send the serum.

The skin test gave a much higher percentage of positive results and was very easy to carry out. The test was usually done with hydatid fluid that had been passed through a filter candle to remove any bacteria that might have gained access. They had found, however, that hydatid fluid from a cyst in the sheep, filtered through sterile filter paper and treated with from 0·5% to 1% of chloroform gave satisfactory results. It was possible, therefore, for country hospitals not possessing filter candles to prepare their own fluid and keep a supply always on hand. In this case it was necessary to use as a control injection sterile normal saline solution containing the same concentration of chloroform as the hydatid fluid.

Dr. Bull added that there were just a few other remarks he might make before concluding. He would like to remind his hearers of the value of the sterile throat swab as a means of collecting specimens of pus and discharges for bacteriological examination. It was necessary for the specimen to be delivered to the laboratory as early as possible before the material had dried. When this was not possible the swab should be rubbed over the surface of a tube of culture medium, a few tubes of which could always be kept on hand and the culture forwarded. This procedure could be used in most cases, but should never be used for the detection of gonococci in pus as mentioned above.

There was one other small matter that might be mentioned and that was the necessity for repeated examinations in many cases. Too much reliance should not be placed on the result of a single examination. This was particularly true with regard to the examination of sputum for the presence of tubercle bacilli. Recently they had examined the sputum from a patient twenty times before they were successful in demonstrating the presence of tubercle bacilli. In concluding he would like to impress on members the value of having specimens examined merely for the purpose of a confirmation of diagnosis if for nothing else. This was particularly true in the case of tissues removed for neoplastic changes or suspected malignant disease. In his opinion every tumour removed should be submitted to histological examination in spite of the fact that the diagnosis was perfectly clear. If this were done, on occasion they would get a surprise, but more important, it made the investigation of the case complete.

PROFESSOR J. B. CLELAND said that Dr. L. G. J. Mackey, in *The British Medical Journal* of January 30, 1926, page 211, had contributed an excellent article entitled "Common Sense." A subleader well worth reading, appearing in the same issue, was called "Wholesale Pathology." One might take these two articles as the text on which hung the matter he had to present and the demonstrations set forth that evening. They should apply common sense in their dealings with the laboratory and the laboratory should exercise common sense in its reports to practitioners. The first common sense point, one so obvious that it should hardly require emphasizing, was that the laboratory must be considered merely as an aid in coming to a correct diagnosis and must not be expected to be alone responsible for the establishment of the diagnosis, except in special cases. In every case a careful inquiry into the history and the fullest clinical examination was more necessary today than ever before in the history of medical science, owing to there being a number of slovenly short cuts to hasty diagnosis. During the course of such a clinical and medical inquiry into the case various points might arise which might necessitate laboratory examinations. The clinical examination should in many cases anticipate the findings in the laboratory, the latter merely confirming on scientific grounds the clinical inferences. Thus by bedside methods the clinician might ascertain that urine which had just been passed by a patient contained pus cells, was acid and was also definitely turbid when passed, the turbidity not being due to the pus cells alone. He drew the inference that he was dealing with coliform bacilluria probably due to a pyelitis. He submitted a catheter specimen to the labortory, seeing that it reached the laboratory immediately after it had been passed, and he awaited the almost certain report from the laboratory that pus cells were present microscopically and that coliform bacilli had been grown. Whilst every avenue should be fully investigated and if necessary exploited, it was not common sense to hurl material from all kinds of sources into the laboratory hoping that from one or other site something might eventuate to throw light on the case. It was not common sense when sending, for example, a specimen of urine to the laboratory to make the request, regardless of expenditure of time and money, that an examination for everything should be made. They had received in vague terms such requests for a full general examination. Under such circumstances the laboratory worker might feel called upon to make cultures in ordinary media, to hunt for tubercle bacilli, to inoculate a guinea pig, to examine for protein substances, to ascertain the percentage of urea, to search for sugar and make a microscopical examination. The practitioner in submitting the specimen should indicate what particular information he desired. He should also accompany the specimen, whatever be its nature, with a reasonably full account of the case, including the age, sex and history of the patient with a provisional diagnosis and a clear statement, when necessary, of the object of the laboratory examination. It was still better if, in addition to supplying that information, which should be done automatically in writing, he should further consult the professional laboratory worker as to the possibilities of the case. The pathologist was only too glad to receive this information and to discuss the case with the practitioner and was often able to suggest other useful lines of investigation or to advise a modification of the request which the practitioner had made to the laboratory. The practitioner should endeavour to get into close touch with the pathologist just as he endeavoured to obtain by personal interrogation from the specialist whom he has called in, information throwing as much light on the case as possible. It would repay the specialist also to consult directly with the pathologist who on the pathological aspect of the specialist's subject must know in general more than the specialist.

It was becoming more and more clear that a fuller and closer knowledge of pathology and bacteriology as it affected other animals was the most likely avenue by which knowledge of human diseases would be increased and some of the unsettled problems solved. The pathology of disease was the same for animals as for man. Modifications of minor degrees occurred, but the reactions were essentially the same. The best pathologists were likely to be those who had made acquaintance with both branches, if separate branches they could be called. At the laboratory at the Adelaide Hospital they were in the fortunate position of having opportunities of conducting work into comparative pathology. They wanted to see in the future a still closer application of the study of diseases of animals to the problems of man. In this way perchance the explanation of the toxæmias of pregnancy might be unravelled. They must realize more and more fully that there was not one pathology affecting animals and another pathology concerned with human beings. The pathologist should know no line of distinction between the two. Pathology itself was no respecter between man and lower animals. The recent establishment of a special section in the Royal Society of Medicine drove this truth further home. Members of the medical profession in Adelaide had a singularly fortunate experience in having available for consultation a pathologist as skilled as Dr. Bull both in animal disease and those of human beings.

Malignant Endocarditis.

Professor Cleland also showed a series of specimens which had been obtained at *post mortem* examination in recent cases of malignant endocarditis.

The first specimen was one of subacute malignant endocarditis of the aortic valve with multiple minute infarctions in the skin and viscera including the intestines and with very speckled kidneys.

The patient, a male, aged thirty-one years, had been admitted under the care of Dr. W. Ray on October 7, 1925, and had died on April 22, 1926. Towards the end of his illness the patient had had a to and fro aortic murmur, a temperature elevated each night, frequent headaches, petechiæ on the hands and complete optic atrophy of the left eye. For a week before death he had had diarrhœa and abdominal pain. Blood cultures made on February 17, March 15 and April 9, 1926, had yielded no growth. Those made on March 4 and 8 had yielded a large Gram-positive bacillus.

At the autopsy showers of petechiæ had been found on the right side, more especially on the trunk and limbs. The heart was somewhat enlarged, weighing three hundred and ninety grammes (thirteen ounces) and the aortic valve was occupied by a number of low fibrinous looking vegetations with the presence of apparently only two aortic cusps which were retracted and had flabby and eroded edges. Both cusps were irregularly perforated. The vegetations extended downwards slightly on to the upper part of the septal wall and towards the mitral valve and in one place there was slight extension to the aorta above one of the sinuses. Infarction, probably due to minute emboli, had evidently occurred into the lower part of the ileum, as for a distance of 12·5 centimetres (five inches) just above the caecum the mesenteric attachment of the ileum manifested a diffuse hæmorrhagic splash. About twelve centimetres (five inches) above this infarcted area was a portion five centimetres (two inches) in extent in which the whole circumference of the gut contained linear hæmorrhagic streaks. Sixty centimetres higher up was a shot-sized hæmorrhagic speck and another was present in the fat of the mesocolon. The lungs were speckled with small hæmorrhages, some of which on section appeared fairly extensive, giving rise to some consolidation. The liver was large, weighing 2·3 kilograms (seventy-seven and three-quarter ounces) and was reticulated with small hæmorrhages. The spleen was very much enlarged, weighing 0·8 kilogram (twenty-seven and a quarter ounces), it retained its shape, was moderately firm and dark red and contained two old, nearly absorbed infarcts. The kidneys were very much enlarged, the right weighing three hundred grammes (ten ounces) and the left three hundred and seven grammes (ten and a quarter ounces). The capsules were peeled with a little difficulty, leaving a closely speckled smooth surface. On section the cortex was swollen and heavily speckled with small red areas, the parenchyma between being paler and the medulla congested. *Staphylococcus aureus* had been grown from the spleen and from a vegetation. Microscopically the kidneys manifested a focal glomerulo-nephritis.

The second specimen was one of malignant streptococcal endocarditis of the aortic valve. A male patient, aged forty-eight years, under the care of Dr. W. Ray, had complained of pains in the back radiating to the buttocks, swelling of the legs, shortness of breath, cough and loss of appetite and weight. His pulse had been regular, but had manifested extra systoles. The patient had been in hospital for two months and had had a swinging temperature for the first nine days. Aortic, diastolic and systolic murmurs had been present. Streptococci had been recovered by blood culture, but only grew in the presence of blood and failed to grow in subcultures.

At the autopsy large soft vegetations had been found hanging from the aortic cusps and extending to the endocardium below. The spleen was scarcely enlarged and had a small old infarct. There was in each kidney a nearly absorbed infarct. There was a small patch of secondary pneumonia in the right lung.

The third specimen was one of malignant endocarditis of the mitral valve. A male patient, aged twenty years, under the care of Dr. Cowan, had given a history of rheumatic fever on three occasions. There had been signs of extensive damage to the mitral and aortic valves. The heart was enlarged, weighing 577 grammes (nineteen and a quarter ounces). There were large vegetations on the mitral valve; they extended upwards on to the posterior wall of the auricle. There was a small vegetation on one of the aortic cusps. The spleen was very large, weighing six hundred and thirty grammes (twenty-one ounces). It was rather firm and dark red, with small scattered infarcts in many places. Both kidneys showed the presence of small infarcts. A staphylococcus had been grown from the vegetations, but it was doubtful whether this was the causative agent.

The fourth specimen was one of malignant endocarditis of the mitral valve. A woman, aged forty-nine years, under the care of Dr. Cowan, had been ill about seven days and had been admitted in a cyanosed condition with rapid pulse and respirations. Moist sounds had been audible at both bases. The heart weighed five hundred and forty grammes (eighteen ounces). Projecting from the mitral orifice into the left auricle was a whitish nodular mass the size of a walnut. The left auricular appendix contained a small white adherent *ante mortem* clot. There were no vegetations on the aortic valve. There was a small infarct in the spleen and an infarct in one kidney. Cultures from the valve had been contaminated. No streptococci or staphylococci had been detected.

The fifth specimen consisted of a lesion in the aortic valve, probably the result of a healed submalignant endocarditis. The patient, a male, had died with a lung compressed by an old empyema and pus in the bronchioles. Scars had been present on the penis. In the aortic valve there was scarring and calcification of the adjacent sides of the right anterior and posterior cusps, the rest of the valve being normal. Professor Cleland thought that this lesion might be explained by a small area of submalignant endocarditis from which recovery had taken place with scar formation. There was no sign of syphilitic aortitis.

Polypus of the Colon.

Professor Cleland also showed a polypus which had been recovered at *post mortem* examination of a man, aged seventy-four years, who had died from the after effects of enlargement of the prostate. The polypus had been found in the transverse colon. It was about 1·25 centimetres long and was attached to the wall of the colon by a delicate pedicle 6·5 centimetres long.

Malignant Melanoma of the Vulva.

A specimen of malignant melanoma of the vulva was shown by Professor Cleland. It had been forwarded by

Dr. Swann. The patient, aged sixty years, had given a history of bleeding for two months, but no pain or wasting. Definite anæmia was present. The growth as yet was small.

Undeveloped Testes.

Professor Cleland's next specimen consisted of undeveloped, descended testes from a man of eunuchoid appearance. The patient, a man of seventy-two year, had died from cellulitis and gangrene. At autopsy the body had presented a feminine appearance, being mistaken at first sight for that of a woman. There was practically no pubic hair. The face was perfectly smooth. Both testes were present in the scrotum, but were very small and undeveloped and only the size of peanuts. It was of interest to note that these undeveloped testes had descended into the scrotum.

Pyloric Polypus.

DR. C. T. C. DE CRESPIGNY, D.S.O., showed a papillomatous polypus acting as a ball valve in the pyloric canal. The specimen had been removed from a woman, III. *para*, aged thirty-five years, who had been three and a half months pregnant and had suffered from vomiting of fifteen months' duration. Symptoms of pyloric obstruction had been present. Dr. Bull had examined the growth and considered it to be innocent.

Raspberry Seeds Passed by the Rectum.

PROFESSOR CLELAND showed a number of raspberry seeds which had been collected by a patient and presented to his medical man for identification probably in the belief that they were gall stones. They showed under the low power of the microscope a characteristic rugose pitted appearance.

Worm Nodule in Corned Beef.

Professor Cleland also showed a portion of corned brisket from a recent Sunday's dinner showing the presence of a small worm nodule due to *Onchocerca gibsoni*. He said that worm nodules were not injurious to health and had been eated with impunity. Their presence, however, was unpleasant and the laity were apt to consider them as tuberculous.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Earlam, Malcolm Sidney Stewart, M.B., Ch.M., 1924 (Univ. Sydney), Devon, City Road, Darlington.
Kite, Miles Gordon, M.B., 1925 (Univ. Sydney), Karralu, Kelso.
Moir, Alistair Edward Martin, M.B., Ch.M., 1926 (Univ. Sydney), Alstonville, Helena Street, Leichhardt.
Sinclair, Bruce Arran, M.B., Ch.M., 1925 (Univ. Sydney), 81, Railway Street, Rockdale.

Medical Societies.

THE SYDNEY HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held in the Maitland Lecture Theatre, Sydney Hospital, on January 21, 1926, DR. T. FIASCHI, D.S.O., the President, in the chair.

Cholelithiasis and an Abnormal Common Bile Duct.

DR. R. BRIDGE showed a male patient, aged sixty years, who had been in good health until eight months previously when he had experienced the first of a series of abdominal crises for which he had sought advice. At the onset of his illness the patient had been very ill and his medical attendant had despaired of his life. During the following eight months the patient had experienced a number of similar attacks of varying grades of severity. The attack had usually been initiated by the appearance of a tumour in the right loin. Shortly after nausea and vomiting had occurred together with severe pain, the pain had been felt through the loin to the back and then all over the back extending as high as the neck on each side. There had been no jaundice and no dyspepsia. From the clinical appearance a diagnosis of intermittent hydronephrosis had been made. Pyelography, however, had proved that the size and shape of the renal pelvis and ureters were normal. The patient had suffered from definite *tabes dorsalis* and it had then been thought that the attacks were renal crises associated with this disease. The tumour had been most elusive. It had formed quickly and in a few hours it would disappear; it had been an "intangible affair" and had appeared to be due to a local spasm of muscle. However, it had been so constant in position and so frequent in occurrence that Dr. Bridge had decided to perform laparotomy.

At operation a most unusual condition had been found. The gall bladder had been thickened and distended with bile and had obviously caused the tumour. The thickening in Dr. Bridge's opinion had not been due to inflammatory disease of the wall of the gall bladder but to an hypertrophy of this organ. It had contained no stones. The common duct had been greatly dilated and contained a stone the size of an olive. Above this there had been a mass of bile mud. The common duct had been incised and the stone and debris had been removed. There had been an immediate escape of much bile and the distended gall bladder had collapsed, demonstrating the patency of the cystic duct. A Hegar dilator had then been passed down the common duct to the duodenum and had disappeared without the exertion of any force for a distance of 12·5 centimetres (five inches). The dilator, however, had not been felt inside the duodenum and it had been obvious that some abnormality had been present. The little finger had then been inserted and at the bottom of the greatly distended duct more stones had been detected. These had been tightly impacted at the opening of the duct into the duodenum and could not be dislodged. Finally, the duodenum had been opened and a finger inserted to steady the stones which were finally removed with a scoop through the opening in the common duct. Three stones had been removed in this way. The Hegar dilator had been passed for a second time. It had traversed the common bile duct for twelve and a half centimetres and its point had been felt with the finger inserted into the opening of the duodenal wall to emerge through an opening in the third part of the duodenum to the left of the vertebral column.

Dr. Bridge said that this unique anatomical peculiarity had excited much interest. The common duct from the junction of the cystic duct to the opening in the third part of the duodenum had been at least fifteen centimetres (six inches) in length. The common duct had passed behind the second part of the duodenum and opened to the left of the spine in the third part of the duodenum. Its relation to the head of the pancreas and the position of the pancreatic duct had not been determined. The patient had made an excellent recovery.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Honorary Treasurer, Professor D. W. Carmalt Jones, of the Australasian Medical Congress (British Medical Association), Dunedin, 1927, is about to visit Sydney, Melbourne and Adelaide. He hopes to meet a large number of medical practitioners interested in the work of the second session of Congress.

The following information is published at the request of the Executive Committee of the Second Session of the Australasian Medical Congress (British Medical Association), Dunedin, 1927.

Accommodation.

The hotel accommodation in Dunedin is not extensive. Those who have enrolled as members of Congress are advised to secure rooms for the duration of the session as soon as possible in order to avoid disappointment. The following charges are made for accommodation for one person each day:

Hotels.

Grand Hotel, from one pound.
Excelsior Hotel, one pound.
Wains Hotel, one pound.
City Hotel, eighteen shillings to one pound for single rooms; eighteen shillings per person for double rooms.
Oban Hotel, fifteen shillings (the majority of the rooms are double).
Criterion Hotel, twelve shillings.

Boarding Houses.

Onslow House, Queen's Drive (about two miles on the tram car route from Dunedin), twelve shillings and six pence and fifteen shillings.
Leith House, George Street (about one mile from the University where Congress will be held), ten shillings.
Ballymena House, High Street, ten shillings.
Applications for rooms should be made to the manager of the hotel or boarding house.

Residential Colleges.

Owing to the limited hotel accommodation in Dunedin special arrangements can be made for visitors to stay at certain of the residential colleges of the University. This accommodation, however, will be available only if a sufficient number of applications are sent in at once to Dr. Russell Ritchie, George Street, Dunedin. The tariff is five guineas per person for the period from February 1 to February 10, 1927. Luncheon will not be provided.

Travelling Facilities.

The Union Steam Ship Company have advised that a steamer will leave Melbourne approximately every three weeks. Arrangements will be made for this steamer to call at Milford Sound on the journey to New Zealand and on the return to Melbourne.

The following ships will sail between Sydney and New Zealand on or about the dates indicated:

Steamship *Niagara* will leave Sydney for Auckland on January 13, 1827.
Steamship *Maunganui* will leave Sydney for Wellington on January 14, 1827.
Steamship *Ulimaroa* will leave Sydney for Auckland on January 21, 1927.
Steamship *Makura* will leave Sydney for Wellington on January 27, 1927.
Steamship *Marama* will leave Sydney for Wellington on January 28, 1927.
Steamship *Ulimaroa* will leave Wellington for Sydney on February 11, 1927.
Steamship *Marama* will leave Auckland for Sydney on February 18, 1927.
Steamship *Maunganui* will leave Wellington for Sydney on February 18, 1927.

Correspondence.

HOSPITAL PRACTICE.

SIR: Recently I sent a patient into a general hospital to have an operation for acute cholecystitis; after some weeks he returned apparently cured, except for a slight sinus in the wound. This sinus continued to discharge; being dissatisfied, I sent him back to the hospital to interview the surgeon who informed him that the primary operation had been incomplete. Such information as I gleaned about the patient I had to seek myself and at no time was I informed that a second operation was necessary.

Such occurrences as this are not isolated experiences; in fact similar experiences have occurred when I have sent patients to the honorary outdoor consultants; in these cases diagnosis and suggested treatment have to be obtained either from the patients (a most unsatisfactory procedure) or else by telephoning the hospital, which generally is more unsatisfactory.

To my mind in these cases the hospital stands in the position as a consultant to the medical attendant. If I send a patient to a consultant privately, I expect and usually receive a memo. advising me of his opinion of the disease and treatment; if I did not, I would consider him, as I now consider the hospitals, unethical and discourteous.

Whilst senior resident in one of the hospitals in England, it was the custom to acquaint outside practitioners by post of the diagnosis and suggested treatment of patients sent to the outdoor and of the treatment performed and conditions found in the indoor patients.

I trust that some such system could be adopted here to the great advantage not only of the medical attendant, but of the general public.

Yours, etc.,

Lane Cove Road, C. H. JAEDE.
Pymble, New South Wales,
May 12, 1926.

Obituary.

JOHN BRYDGES MASON.

WE regret to announce the death of Dr. John Brydges Mason which occurred at Launceston, Tasmania, on May 22, 1926.

GEORGE LOCKWOOD LAYCOCK.

WE announce with regret the death of Dr. George Lockwood Laycock which occurred at Windsor, Victoria, on May 24, 1926.

HAROLD MAUND EVANS.

WE regret to announce the death of Dr. Harold Maund Evans which occurred at Adelaide, South Australia, on May 21, 1926.

THE MEDICAL DIRECTORY.

CIRCULARS containing forms to be filled in for the compilation of the Medical Directory have been addressed to every medical practitioner in Australasia. Those who did not reply to the first invitation, received a second circular. There are still many who have not entered the details required on the forms and returned them to The Printing House, Seamer Street, Glebe, New South Wales. The form has been reproduced in this issue and will be found in the Advertiser. Members who have not already sent in their forms, are earnestly requested to tear out the page, to fill in the necessary details and to transmit it to this address as soon as possible.

PERSONAL.

EARLY in April of this year Dr. G. H. Taylor resigned the position of Medical Officer to the Railway and Tramway Department of the New South Wales Government after a period of eighteen years. Dr. Taylor has had a long and remarkable career in the service of the New South Wales Government. As Pathologist to the Department of Public Health and as Medical Superintendent he has proved himself to be both an original thinker and a highly competent

observer. We have had occasion within the last year or two to refer to his work on leprosy. Several years ago he was able to show that the diagnosis of early leprous lesions was possible at a stage when Hansen's bacilli could not be demonstrated and that the disease was commoner than it was supposed to be. His doctrine has received ample confirmation. It has been proved beyond reasonable doubt that in countries where leprosy is endemic, many persons suffer from the disease in a mild form which is not readily detected. Since the disease is a self-healing one, the majority of these people recover spontaneously. That they are a source of possible infection has also been demonstrated. When Dr. Taylor propounded his ideas on this subject, he received nothing but discouragement and opposition. His work on colour vision and the detection of the various forms of colour blindness is well known and has proved to be of inestimable value in the railway and tramway service.

JOHN IRVINE HUNTER MEMORIAL FUND.

THE following subscriptions have been received by the Honorary Treasurer of the John Irvine Hunter Memorial Fund:

Amount previously acknowledged	£2,087	2	2
Drs. James and McIntyre Sinclair	100	0	0
Dr. W. C. B. Harvey	10	10	0
Sydney University Medical Society	10	10	0
The Women Undergraduates' Association	7	10	10
Dr. Charles R. Smith	5	5	6
Professor Peden	5	5	0
Dr. Dudley Henry	5	5	0
Dr. F. N. Waddell	5	5	0
Dr. R. A. Rankine	3	3	0
Dr. B. T. Edye	2	2	0
Dr. V. P. Mondon	2	2	0
Dr. W. D. Hawke	1	1	0
Miss A. Stewart	0	10	6
	£2,245	12	0

Medical Appointments.

Dr. Tristram Essx Young Holcombe (B.M.A.) has been appointed Government Medical Officer at Stroud, New South Wales.

* * * *

Dr. Victor H. L. Dunn (B.M.A.) has been appointed a Commissioner of the Lancefield Waterworks Trust, Victoria.

Books Received.

HANDBOOK OF DISEASES OF THE RECTUM, by Louis J. Hirschman, M.D., F.A.C.S.; Fourth Edition, Revised and Rewritten; 1926. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 402, with illustrations. Price: $6.50 net.

MODERN METHODS OF AMPUTATION, by Thomas G. Orr, A.B., M.D., F.A.C.S.; 1926. St. Louis: The C. V. Mosby Company. Royal 8vo., pp. 117, with illustrations. Price: $3.50 net.

THERAPEUTICS, MATERIA MEDICA AND PHARMACY, by Samuel O. L. Potter, A.M., M.D., M.R.C.P. (London); Fourteenth Edition, Revised by R. J. E. Scott, M.A., B.C.L., M.D.; 1926. Philadelphia: P. Blakiston's Son & Company. Royal 8vo., pp. 986.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xxiv.

ST. JOSEPH'S HOSPITAL, AUBURN: Honorary Radiographer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital. Cook District Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

JUNE 15.—Tasmanian Branch, B.M.A.: Council.
JUNE 15.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 16.—Western Australian Branch, B.M.A.: Branch.
JUNE 16.—Victorian Branch, B.M.A.: Council.
JUNE 16.—Central Northern Medical Association, New South Wales.
JUNE 16.—South Sydney Medical Association, New South Wales.
JUNE 16.—Section of Obstetrics and Gynæcology, New South Wales.
JUNE 17.—Section of Neurology and Psychiatry, New South Wales.
JUNE 21.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 22.—Illawarra Suburbs Medical Association, New South Wales.
JUNE 23.—Victorian Branch, B.M.A.: Council.
JUNE 23.—Western Medical Association (Forbes), New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.